PHYTOPATHOLOGY

VOLUME III, 1913

PHYTOPATHOLOGY

OFFICIAL ORGAN OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

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BUSINESS MANAGER

DONALD REDDICK

VOLUME III

JANUARY–DECEMBER, 1913

WITH 28 PLATES, AND 32 TEXT FIGURES

PUBLISHED FOR THE SOCIETY
WILLIAMS & WILKINS COMPANY
BALTIMORE, MD.

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HARRY MARSHALL WARD

PHYTOPATHOLOGY

VOLUME III NUMBER 1

FEBRUARY, 1913

HARRY MARSHALL WARD
(1854–1906)

E. M. FREEMAN

WITH PORTRAIT, PLATE I

While Americans are prone to look to Germany as the kind mother of the botanical sciences none will deny that England has contributed at least her fair share of able men and advanced ideas. Nehemiah Grew, Robert Brown, Sir William Hooker, Lindley and Charles Darwin are names that any nation would present with pardonable pride. But in the closing quarter of the last, and in the few past years of the present century botanical England has awakened to a new life, remarkably fruitful of men and research, until today her laboratories and schools command a most wholesome respect in all quarters of the globe.

Harry Marshall Ward was a factor of no small importance in this renaissance. England owes him a deep debt of gratitude, but the science of pathology also acknowledges his contributions not only of research but of the spirit and power which he imparted to the botanical movement.

To the average American the career of this little giant of pathology offers a fascinating story, particularly appealing to those who are more wont to associate such triumphs over natural difficulties with the greater latitudes of a youthful democracy rather than with a staid England. Ward's early career was fraught with difficulties, and his education and training were well and hard earned. Indomitable courage and an endless capacity for work made possible not only his early training, but constituted the basis of his future research. He apparently delighted in such stupendous tasks as the bacterial flora of the Thames, and dug deep into every nook and crevice of any field which he had started to explore. Thoroughness and detail he developed to a remarkable degree, stimulating and eminently sound in its effect on his own research as well as on that of his pupils, but inclining his presentation perhaps to undue length of description. In spite of such detail his teachings and numerous published works are marked by clearness and logical exposition. He combined with this love of detail an extraordinary breadth of knowledge and interest. No important field

of botanical research escaped him, and his powers of assimilation and correlation were astonishing.

Chance perhaps led him chiefly into the fields of pathology. His early work on the coffee rust of Ceylon seems to have given a permanent bent to his botanical career. From 1881, the time of his return from Ceylon, to 1888, Ward's contributions dealt with a large range of mycological subjects, including important results on the tubercle organisms of leguminous plants. He had previously studied with Sachs at Würzburg, and later with De Bary at Strasburg. These continental visits, while not extensive in time, were fruitful in inspiration and influence on his future work. In 1888 he published his paper On a Lily Disease in which he seems to have become fairly launched in what was probably the main problem of his life work, the problem of parasitism. His attention was naturally attracted to any association of plants in intimate relations of nutrition, and his paper on the Ginger Beer Plant opened the way to new conceptions of fungous nutrition. His problems of plant parasitism were now interrupted by that tremendous and almost appalling task the bacterial flora of the Thames, which work was at least remarkable for the wealth of laborious detail.

Upon his appointment to the chair of botany at Cambridge, in 1895, Ward took up again the question of parasitism and attacked the problems presented in the cereal rusts. While he was engaged in this work Eriksson startled the mycological world with his theory of mycoplasm. With characteristic keenness and thoroughness Ward subjected the mycoplasm theory to rigid laboratory and field tests. He was soon convinced of the fallacy of the theory and at once set about with his usual vigor to combat the hypothesis. While it is undoubtedly true that the controversy called forth productive research in many ways, yet it is to be regretted that the continuity of Ward's work was somewhat impaired by the controversial demands which were out of proportion to the value of the results. That his contentions in this controversy were well established few pathologists question.

Ward's last important work carried into further fields the problems of rust parasitism, especially with the brome rusts. His results, though far from satisfactorily completed as far as his own views were concerned, have been beacon lights of research along the most important road of the quest for the true meaning of parasitism, immunity and susceptibility.

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THE MIGRATION OF BACILLUS AMYLOVORUS IN THE HOST TISSUES

FREDA M. BACHMANN

WITH PLATES II AND III AND TWO TEXT FIGURES

SUMMARY OF PREVIOUS STUDIES

The little study which has been given to the migration of *Bacillus amylovorus* in the tissues of the host has resulted in quite different conclusions on the part of the several investigators who have taken up the subject. A knowledge of the exact manner of migration of the organisms is of equal interest to the horticulturist in its practical application to methods of pruning for the extermination of the disease, and to the general student of biological problems in the relation of the parasite to the cells of the host tissues. The problem was suggested to me by Prof. L. R. Jones under whose direction all of the work has been done. I wish to acknowledge my indebtedness to him for helpful criticism and suggestions throughout the progress of the work.

As is well known, *Bacillus amylovorus*, as the cause of blight in pear, quince and apple trees, was discovered by Burrill (2) in 1877. More recently it has been found to be the causal organism in the blight of plum (Jones, 4) and apricot (Paddock, 6). Burrill's conclusions concerning the relations of the organism to the tissues may be summarized as follows: the bacteria produce butyric fermentation of the stored carbonaceous compounds in the cells, especially in those of the bark outside the fibrous inner layer; the most conspicuous change in the tissues is the disappearance of stored starch; the cell walls are not dissolved or altered except the staining by oxygenized material in later stages of the disease; the protoplasm of young cells remains until death takes place; in older cells the thin protoplasmic lining of cell walls can be seen after the starch has disappeared; bacteria swarm in cells absolutely closed, with no observable corroding of the cellulose wall whatever. To account for the entrance of bacteria into such closed cells, Burrill suggested that, if we accept Nägeli's theory of molecular construction of the cell wall, the bacteria may be so plastic that, like amoebae, they may creep through the spaces between the molecules, or in case they are smaller than the molecular openings they may pass through these from cell to cell. Burrill further observed that in very young tissues all parts except the epidermis are equally affected,

but in older limbs the chlorophyll-bearing parenchyma of the bark is the first and usually the chief seat of the disease; the bast is not affected and the cambium may retain its vitality when all outside has perished; the xylem may be stained by the ascending water colored in its upward passage through the dying and brown parts.

By using the sterile filtrate of a strong solution of blighted tissue for inoculating green pears, Arthur (1) found in 1885 that the disease was produced by the organisms and not by their products.

In 1895 Waite (9) records that the inner bark and cambium layers are killed but that the bacteria rarely kill the leaves, only occasionally attacking the stems and midribs. The leaves die after the branch is killed. As to the rate of progress of the disease, Waite found that in one day it rarely extends farther than 2 or 3 inches from the point of attack, but occasionally travels as much as 1 foot. Warm moist weather with frequent showers favor blight while cool sunny dry weather hinders it and very dry weather may check it entirely. In old dried bark where the disease has stopped the bacteria have all died and disappeared.

Whetzel and Stewart (10) state that the bacteria do not travel through the sap tubes but slowly work their way through and between the cells of the bark. Shortly after the publication of their observations, D. H. Jones (5) published a bulletin in which quite different views are held. He figures the diseased pedicel of a young apple in which he says the cells are "surcharged with *B. amylovorus*." He also finds the cells of apple fruit filled with bacteria. He states that "the bacillus lives in the cells of the inner bark, feeds on the cell contents and, as it develops and multiplies, passes along from cell to cell, destroying the tissue as it progresses. It may travel down the twig at the rate of from 0.25 inch to 2 inches a day."

In view of these diverse opinions, it seemed worth while to make a careful examination into the histological relations of host and parasite. In my studies I have inoculated blossoms of pear, water-shoots of apple, young pear seedlings, young shoots of pear and plum and fruits of pear and apple.

INOCULATIONS OF PEAR FLOWERS

Pear flowers were inoculated by placing a drop of a bouillon culture of *B. amylovorus*, in the outer calyx cup. As is well known, it is not necessary to make any mechanical injury to secure infection, for the bacteria readily gain entrance into the tissues through the nectary. The infected calyx cup and pedicel of the flowers were later fixed in various fixing fluids, by far the best results being obtained with picro-formol. Normal tissue for comparison was fixed in exactly the same way at the same time. Sections were cut 10 microns thick.

In these sections there can be no doubt at all as to the path of migration of the bacteria. The cells are not so large but that much of each cell is seen in the section. The walls are very thin and the intercellular spaces large. The slimy cytoplasm forms a thin, peripheral somewhat granular layer. The granules in the cytoplasm are so very minute that it is not possible to confuse them with microörganisms. In inoculated tissue the intercellular spaces contain few to many bacteria (text fig. 1).

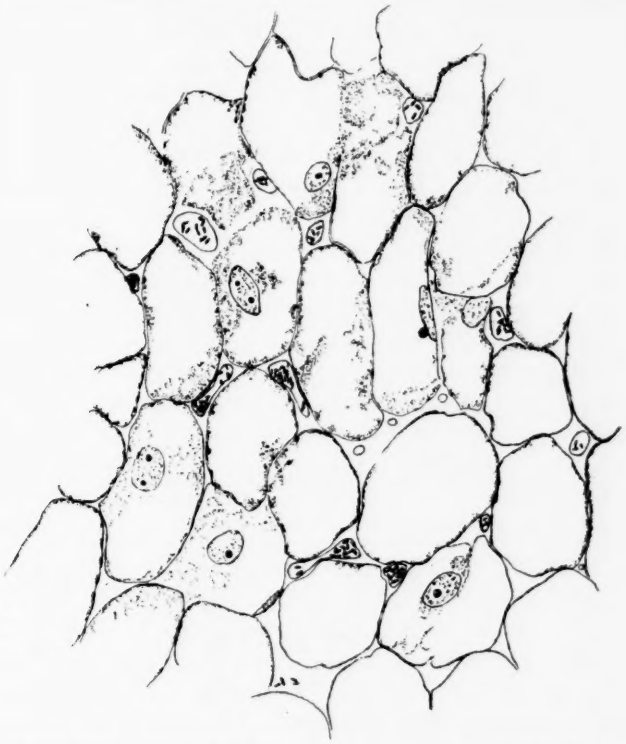

FIG. 1. Cross section of pedicel of inoculated pear flower showing bacteria surrounded by film of liquid in the intercellular spaces. × 580.

Bacteria may be found in at least one-half of the intercellular spaces at a time when the cell contents are in no way disorganized. There may be at this time some indication of plasmolysis but the slimy layer of cytoplasm is still next to the cell wall and the only difference between these cells and those of the normal tissue is that they are less turgid.

The bacteria are surrounded by a film of liquid which it seems likely has been drawn out from the cell sap. In the parenchymatous tissue they are never found within the cells in early stages of infection and cer-

tainly migrate entirely between the cells. In somewhat later stages of infection there are sometimes broken places in the cell walls, but no tendency as yet for the bacteria to enter the cells. It is possible that these broken places are the result of a chemical dissolution of the cell wall by substances secreted by the bacteria. However, we should then expect the wall gradually to become thinner until large openings resulted, but this is not the case. The appearance of these torn places suggests, rather, that the break is due to a mechanical rupture. It is not impossible that some chemical dissolution may first weaken the wall, which is then easily torn. Even when the tissue has become much shrunken and the cells distorted the bacteria are found, with few exceptions, only in the intercellular spaces. Here they are often crowded so that the group stains very deeply. In very late stages of infection the cells are completely plasmolyzed and the walls more or less broken. The dead tissue occurs in masses with large open spaces between. These masses contain many bacteria. Often, when the broken cell wall is not collapsed, the bacteria fill the space within. The protoplast in every case becomes so much plasmolyzed that it forms a dense, often quite homogeneous, mass. I have observed no tendency for the bacteria to invade the protoplast and it is very evident that they are not found abundantly in any of the cells before the death of the latter.

INOCULATION OF SHOOTS OF APPLE, PEAR AND PLUM

For this study water shoots of apple, seedling pears or young shoots of apple not over 3 inches long and young shoots of plum were used. The inoculations of pear and plum were made on greenhouse plants. In the case of apple, the water shoots were brought into the house, inoculated and then placed in a damp chamber. The same method of inoculation was used throughout, viz:—a drop of a bouillon culture of *B. amylovorus* was placed on the side of the stem from one to a few inches from the tip of the shoot and then by means of a sterile needle the epidermis was ruptured within the drop. Picro-formol gave the best results as a fixing fluid. Normal tissue for comparison was always fixed at the same time. Most of these sections were also cut about 10 microns thick.

In unstained hand sections of living shoots after inoculation I have found that a plasmolysis of the cells of the cortex, and to some extent of the pith cells, is one of the first indications of disease resulting from the bacterial invasion. In stained sections it is evident that the large intercellular spaces of the cortex provide a ready path for the migration of the organisms. These spaces, which are frequently as large as the cells, are often seen packed with bacteria (pl. II, fig. 2). Indeed it is

not impossible to mistake these large spaces so filled with deeply staining bacteria for cells, if care is not taken in focusing. In early stages of infection in apple shoots I have found many of these spaces crowded with bacteria when the cells were only partially plasmolyzed and the cell walls seldom broken. The organisms migrate just as readily between the cells of the pith (pl. II, fig. 1) but, since I have sometimes found the pith quite or entirely free of bacteria when all the other parts are infected, I am inclined to think that the organisms do not readily gain entrance to this region unless the wound made by inoculation has extended through the bundle ring. Later, the walls of the cells of cortex and pith sometimes become broken in places so that bacteria may enter the cells. Frequently some of the cell walls are broken and the surrounding ones collapsed so that large open spaces more or less filled with bacteria result.

I have found that the bacteria enter the xylem tubes very readily (pl. III, figs. 1–3). These tubes, in some places in a shoot, are so packed with the organisms that the transmission of sap in such a region seems quite impossible. In a plum shoot inoculated about 5 inches from the tip many of the xylem vessels 3 inches above the point of inoculation were filled with bacteria. Sections only about 1.5 inches above the point of inoculation seem to show fewer bacteria in the vessels. This would suggest that the bacteria entered the xylem some distance above the point of inoculation and then migrated downward in these tubes. The same phenomenon was observed in a seedling pear about 3 inches in height. This was killed and fixed eight days after inoculation. The xylem tubes were found free of bacteria except near the apex of the shoot where in some of the tubes bacteria were in abundance. In another young seedling pear of only 2 inches, also fixed eight days after inoculation, where the point of inoculation was about in the middle of the stem, the xylem tubes both in the upper and lower part of the shoot were full of bacteria. In such exceedingly tender tissues as those of very young pear seedlings it may be that the organisms can penetrate the xylem tubes at any place. However, it is not impossible that here too the organisms may work downward in the stem. Oddly enough some xylem tubes may be filled with the organisms while the adjoining ones are entirely free. Longitudinal sections show this especially well. The parenchyma cells between the xylem tubes are densely filled with granular cytoplasm and are usually entirely free of bacteria. There is apparently no tendency for the organisms to enter these cells.

It is probable that the bacteria in some way destroy or break through the thinner parts of the walls of the tracheids. The thin-walled cells between the xylem become completely plasmolyzed and partially disintegrated so that large open spaces in the xylem are formed (pl. III, fig. 1).

The xylem tubes often appear as loose partly uncoiled spirals (pl. III, fig. 2). When all the xylem elements are filled with bacteria, or completely destroyed, the cambium cells may to all appearances be normal. But these, too, may be destroyed later. By the time the tissues appear blackened the cells are completely plasmolyzed and apparently dead. The petioles of leaves and stems which appear to be in a perfectly healthy condition have been found to be invaded throughout by the organisms. An inoculated seedling pear showed only a narrow streak of blackening on the edge of the stem on the upper two-thirds of the shoot and at the bases of the leaf petioles. The leaves gave no evidence of wilting. When this seedling was sectioned the xylem tubes were found to be full of bacteria in the upper part of the plant; the intercellular spaces of pith and cortex contained many bacteria and in some places the cells were destroyed, leaving relatively large cavities filled with bacteria. A leaf petiole which showed a few bacteria in the xylem and in which the parenchyma cells were badly plasmolyzed, when traced to its origin was found to come from a region where the xylem tubes were completely plugged by the organisms and where many of the cell walls were broken. The cells in the apical region of this seedling were found to be entirely normal while, only a few sections from the tip, bacteria were abundant in the intercellular spaces. Farther away from the tip occasional large areas in the pith had been destroyed. It is very evident from this series of sections that the primary path of the migration of the organisms has been through the intercellular spaces and that the xylem tubes were invaded later.

INOCULATION OF FRUITS

For this work green hard Bartlett pears and Whitney Crab apples were used. The pears were from 1.5 to 2.5 inches long and the apples about 1.5 inches in diameter. The calyx end of the fruit was cut three-fourths across with a sterile knife in the manner indicated in the diagram (text fig. 2) so that a part remained attached. Then with a sterile platinum loop bacteria were transferred from a pure agar or bouillon culture of *Bacillus amylovorus* and smeared over the cut surface. The exudate which appears on old infected fruits was also used for inoculation. The upper portion of the fruit was then pressed rather firmly down so that there would be as little exposure to drying as possible. The entire fruit was then placed in a covered jelly tumbler containing just enough water to submerge the end of the stem. The inoculated fruits were either kept at room temperature or in the refrigerator. The fruits were observed from day to day and results noted as to the production of exudate or as to rotting. A softening of the tissues, with accompanying transparency,

is what I have called fairly well rotted. The exudate appeared first on the cut surface of the fruit and later was exuded in beads on the entire outer surface. The first evidences of rotting showed as quickly in the apples as in the pears.

Small pieces of both inoculated and normal apple and pear were fixed in picro-formol, Merkel's fluid or Flemming's strong solution of chromosmic-acetic acids. Not all of the material has been sectioned but satisfactory results were obtained with both the Merkel and Flemming mixtures. The cells of these green fruits are quite large so that it was found necessary to cut the sections rather thick, preferably about 20μ. *B. amylovorus* stains fairly well with the triple stain but much more readily and more sharply with carbol fuchsin. The latter, either alone or combined with gentian violet and orange G, has also given very good results

FIG. 2. Diagram showing how fruits of apple and pear were cut for inoculation.

in staining the structures of the normal cell. For this reason I have preferred the use of carbol fuchsin instead of safranin.

The great bulk of tissue of these fruits consists of large, very irregular parenchyma cells with abundant intercellular spaces. The cell walls are quite thin but stain deep pink with carbol fuchsin or blue with gentian violet. These parenchyma cells have very large central vacuoles with a thin peripheral, somewhat granular cytoplasmic layer within which is the nucleus, usually flattened somewhat against the cell wall. The nuclei are easily stained and contain one or more deeply staining nucleoles and chromatin distributed throughout in a finely granular condition. This description serves for that parenchyma which composes at least the greater part of the calyx cup. The parenchyma cells which are nearer the center of the fruit contain a great number of granular microsomes which vary somewhat in size. Some of the smaller ones might easily be mistaken for bacteria, the more so since they stain as deeply, as a rule, as do the

bacterial cells. The starch grains which are scattered about in the cells vary in size but they are usually many times larger than the small granules I have described. The parenchyma cells containing these masses of granules are nearer the center of the fruit, and hence near the fibro-vascular tissues. Bunches of sclerenchyma cells are found scattered throughout the pear tissues.

In some cases I have fixed thin longitudinal slices through a half of the fruit so that it is entirely possible to trace the bacteria from the point of inoculation and also to observe all stages of disintegration of the tissues. In the longitudinal sections of pear the well-rotted tissue near the inoculated surface is stained a deeper red than the remaining tissue. This is due to the fact that the cells have lost all turgidity and are for the most part almost entirely collapsed. The content of the cells occurs in irregular masses, very dense in some places, and occasionally granular. It is not possible to say that in a cell which contains a great deal of finely granular material there are absolutely no bacteria at all, but the more I have studied my preparations the more inclined I am to doubt that the bacteria enter these cells to any great extent, if at all.

On the other hand, bacteria may often be found between the cells and on the outer surfaces of some of the cell walls. It might at first appear that the organisms are imbedded in the red-staining material within the cell, but this is easily disproved by the many cross sections of cells which never show the bacteria inside the cells. Frequently, they lie so close to the cell wall that it appears as if they might be at least partially imbedded in it. In even this well-rotted area the walls are for the most part unbroken, although occasionally they are torn. It may be possible that this tearing is at least partially due to sectioning, since in the pear the masses of sclerenchyma tend to tear away from the other tissues. In a few cases I have thought it possible that a few organisms may have entered cells where the walls are torn, but I am very certain that the cells in this much-rotted tissue are at least not packed full of bacteria. My preparations are from tissue which had been inoculated from six to ten days. It may be that in fruits which have been inoculated a much longer time bacteria would be found within the cells. But, in this connection, I have observed that there are fewer organisms in the well-rotted parts than in those parts which show very little or no rotting. In these less-rotted regions the location of the bacteria is the same. Here the cells are less collapsed and the intercellular spaces are larger. These spaces are filled with bacteria (pl. II, fig. 4). In parts of the section still further away from the point of inoculation the cells are more turgid and the bacteria less plentiful and, again, they are only in the intercellular spaces. Pears 2 inches long kept in the ice chest for six days after

inoculation showed at this time bacteria scattered throughout the fruit in the intercellular spaces.

In the inoculated apple fruits which I have sectioned rotting had not progressed to any extent beyond the inoculated surface and organisms were found only very near this surface. However, here too, the path of migration appears to be the same as that in the pear, since I find the bacteria only between the cells and not within them. Those cells which are exactly on the inoculated surface of the section, and which were doubtless torn in cutting the fruit for inoculation, contain bacteria, but besides this I find no bacteria within the cells in these early stages of rotting.

As to the migration of the organisms in green fruits of pear and apple, I conclude that the path of migration is through the intercellular spaces from the region of inoculation to all parts of the fruit. They apparently extract the cell sap from the cells thus causing the death of the latter. Toxic substances, if produced, probably do not precede the advance of the bacteria, since bacteria are found between apparently normal cells. I can find no evidence at all in my sections of pear and apple that the bacteria progress from cell to cell.

CONCLUSIONS

The first evidence of infection in the tissues of fruit or shoot is a transparency around the point of inoculation, followed later by a browning in the same region. From my studies I conclude that this transparency is due to the removal of air from the intercellular spaces, this being replaced by the liquid in which the bacteria live. Doubtless this liquid is cell sap which has been extracted from the cells, thus causing them to lose their turgidity. The cells die, apparently because of a loss of water, although chemical changes in the protoplast may accompany this loss. The substances produced in the metabolism of the organisms are, judging from the microscopic evidence, not at all strongly or quickly toxic in their effect on the cells. This is evident because the organisms are found abundantly between cells which to all appearance are entirely normal. The film of liquid in which the bacteria move is not extracted in such amount that it precedes the bacteria to any extent. My conclusions on the non-toxicity of the bacterial products on the cells of the host are in agreement with some experiments of Arthur (1) who found that solutions in which the organisms were grown, when filtered and placed in green fruits, resulted in no rotting at all. It seems to me that at least all of the first changes in the cells which result from infection may be attributed wholly to a loss of water. However, it is possible that the disintegration of the tissues may result in poisonous products which later hasten the death of other

cells. The relations of the bacteria to the host is much like that of the rust *Uromyces pisi* to its host *Euphorbia cyparissias*. Tischler (8) found that the mycelium here never enters cells which are full of protoplasm such as those in the growing regions but readily sends haustoria into those cells which have become vacuolated. The mycelium grows between and among the cells and in the xylem tubes. Here too, as I have found, some of the xylem tubes are full of mycelium while others adjoining are entirely free from it. Another point of likeness is that the mycelium in the older infected parts is dead and that it lives only near the growing regions.

As to the rate at which the bacteria may migrate I have no data. Watershoots of apple forty-eight hours after inoculation were fixed and when sectioned the organisms were found one-half inch from the point of inoculation. At this time there was only a slight transparency around the wound. I have repeatedly found that tissues which are to all appearances healthy contain many bacteria in the intercellular spaces.

Burrill (3) concluded that the bacteria feed on the carbonaceous material in the cells. In the cells of pear fruits there appears to be somewhat less granular material in the tissues which are diseased. In apple I have not observed a diminution in the amount of starch in the cells. The cellulose walls are not digested early, although it seems that digestion in some portions of the walls may have occurred to cause the broken places. If there is a process of cellulose digestion it certainly goes on very slowly and not uniformly over all surfaces. However, since the bacteria are most abundant in the intercellular spaces it is possible that we have here the result of what Smith (7) terms a mass action of bacteria, i.e., a result which can only be brought about by a large number of organisms. It seems possible to explain the broken walls on a purely physical basis. There is certainly a very great pressure exerted to cause the exudation of so much slime on the surface of the twigs. The cortical cells of the bark are very close together and some force must be exerted to cause them to tear apart. It may be that the osmotic pressure of the substance in which the bacteria are found is sufficiently great to rupture the cell walls. Evidence of such pressure is found in the large cavities which are formed in the xylem, cortex or pith. These cavities are frequently rounded or oval and suggest an equal pressure in all directions. The result of a process of enzymic solution should be the gradual dissolution of the entire wall and not merely a tear at one place. There probably is such digestion of the walls to some extent but osmotic pressure is, in our judgment, a more important factor in their final rupture.

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EXPLANATION OF PLATES

(All figures were drawn with the aid of a camera lucida)

PLATE II

FIG. 1. Longitudinal section of pith from inoculated shoot of apple showing organisms between the cells. × 750.

FIG. 2. Transverse section of cortex from inoculated shoot of apple, showing cells plasmolyzed and organisms for the most part between the cells. × 450.

FIG. 3. Surface view of cortical cell walls from apple shoot, showing organisms on the outer surface of the walls but not in the cells. Some of the walls ruptured. Breaking of walls probably due to great osmotic pressure. × 750.

FIG. 4. Section through inoculated pear fruit. Cells plasmolyzed and organisms in intercellular spaces. × 450.

PLATE III

FIG. 1. Cross section of inoculated apple shoot, showing organisms in the xylem tubes, large cavity found in xylem and pith cells plasmolyzed. × 750.

FIG. 2. Same as figure 1. Part of the walls of xylem tubes destroyed. × 750.

FIG. 3. Cross section of inoculated apple shoot showing earlier stages of infection than figures 1 and 2. Organism in the xylem tubes and to all appearance between the thicker parts of the wall. × 750.

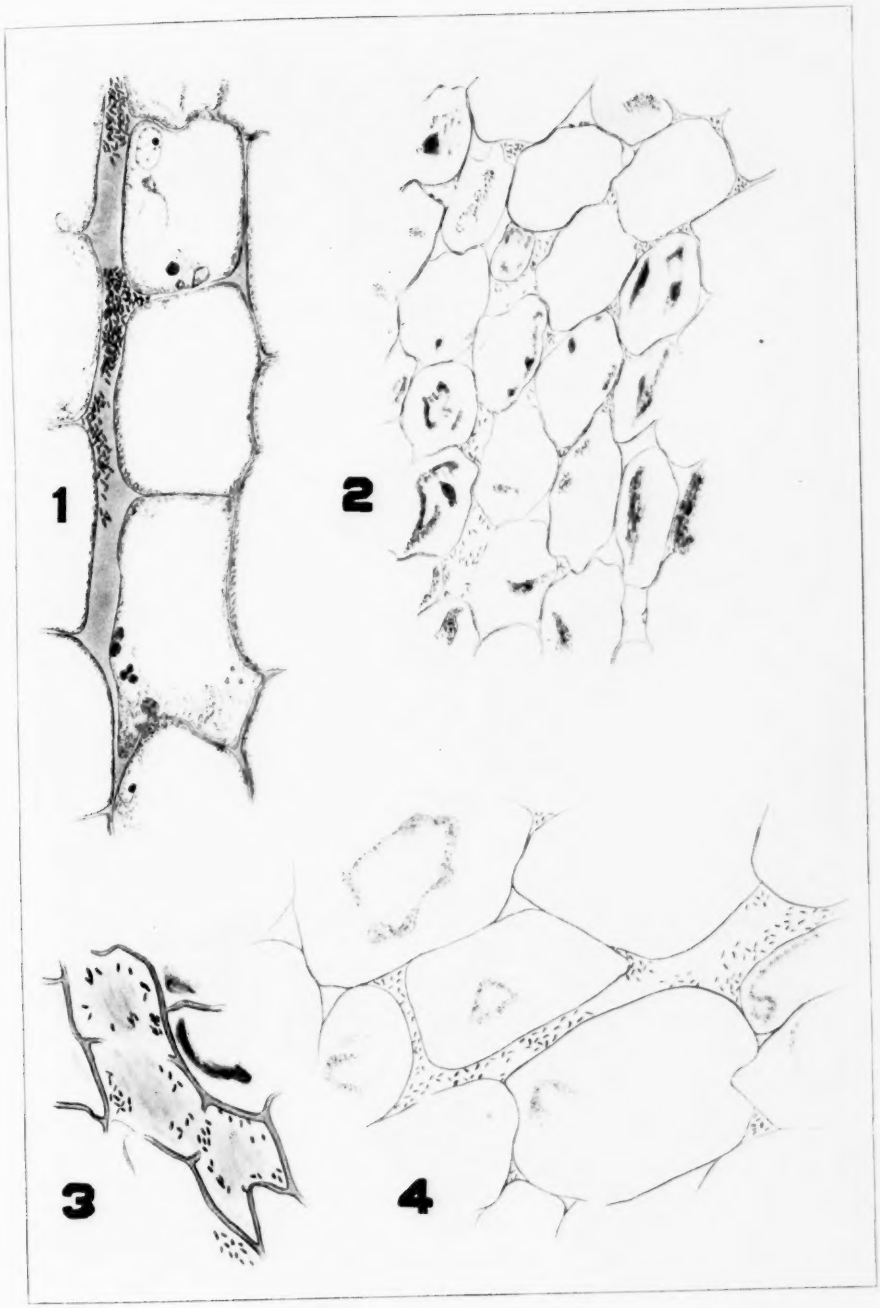

PLATE II.—BACILLUS AMYLOVORUS

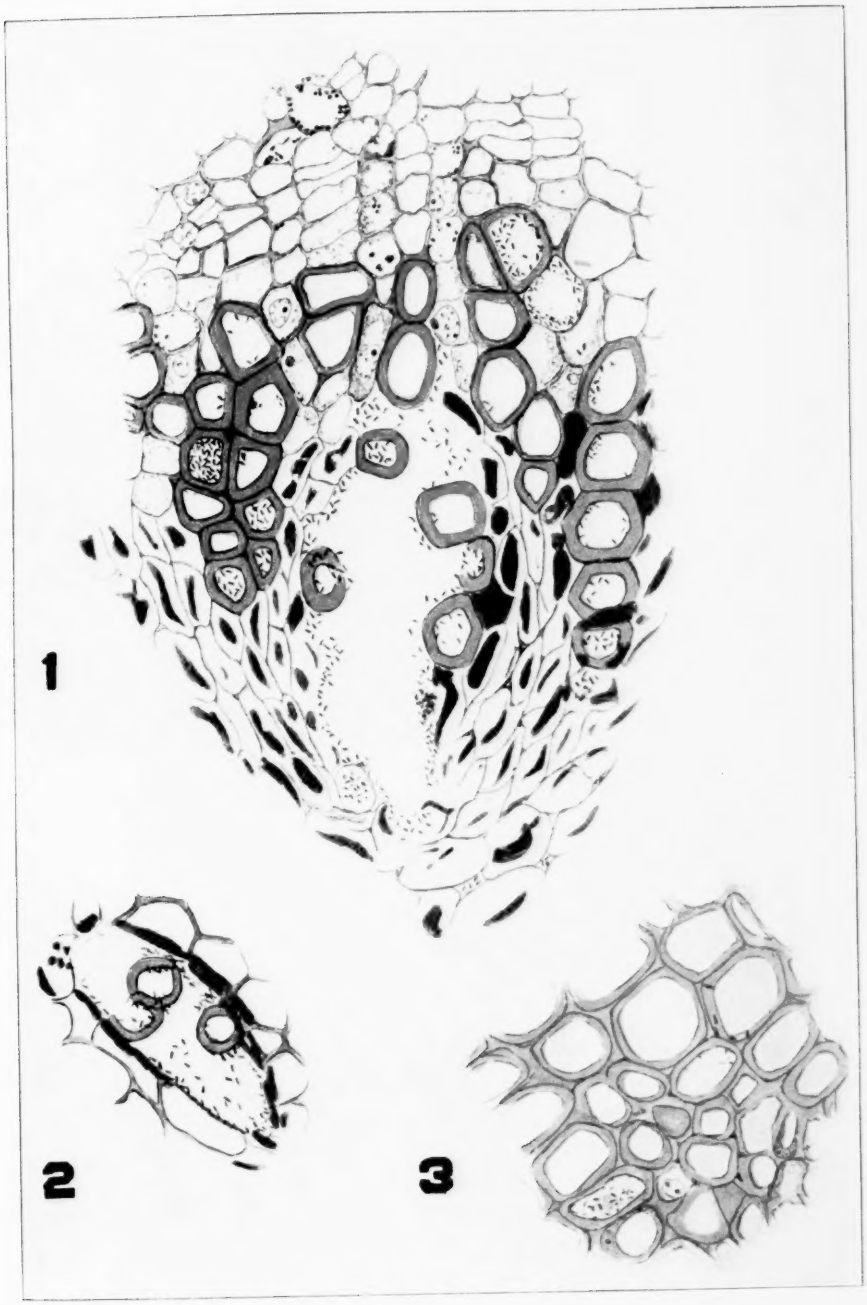

PLATE III.—BACILLUS AMYLOVORUS

NOTES ON SOME WESTERN UREDINEAE WHICH ATTACK FOREST TREES. II[1]

GEORGE GRANT HEDGCOCK

The following paper is a continuation of notes on observations made on field trips in the west by the writer.

1. PERIDERMIUM FILAMENTOSUM Peck

This fungus, in an overmature condition, was found in abundance on a number of trees of *Pinus ponderosa* Laws. near Rollinsville, Colorado, August, 1912. Plants of Castilleja in the immediate vicinity were badly injured by its alternate stage, *Cronartium filamentosum* (Peck) Hedgc.[2] No other species of Peridermium was found in the vicinity.

On September 5, 1912, a number of trees of the same species were found diseased with this Peridermium in the forest on the Fremont Experiment Station of the Forest Service on Pikes Peak, Colorado. Most of these were badly diseased and decadent, although they were young trees not over fifty years of age. In one instance freshly-fruiting aecia of the fungus were found. This is unusual, for the fungus, as a rule, fruits during the latter part of June and the first of July. As at Rollinsville, the Castilleja plants in the immediate vicinity were badly diseased with *Cronartium filamentosum*. No *Peridermium harknessii* Moore was found in this locality.

It might be well at this point to state that *Peridermium filamentosum* is the most destructive species of Peridermium that has been observed in the west. The first time it fruits abundantly it apparently kills the limb affected, due to the drying out of the tissues ruptured by the large aecia. On the one hand, this feature tends to limit the ravages of the fungus, since the fungus itself is thereby killed, but this is offset, on the other hand, by the fact that the fungus is apparently able to grow along the cambial layer from one twig or limb around a fork into another, thus spreading the disease without reinoculation.

Where close grazing by cattle or sheep occurs, which is now usual in western forests, the Castilleja plants are browsed so closely that little

[1] Published by permission of the Secretary of Agriculture. The first paper was read at the 1911 session of the Phytopathological Society in Washington, and published in Mycologia **4**: 141–147. May, 1912.

[2] See Phytopathology **2**: 176–177. 1912.

or no *Cronartium filamentosum* is found and the spread of the fungus is held in check. This is fortunate, indeed, and so long as this condition exists, little is to be feared from this rust in closely grazed forests.

2. PERIDERMIUM HARKNESSII Moore

This species of Peridermium was not found in abundance in any locality visited this year. No good evidence has been found to support the assumption made in my previous paper that this fungus may have a telial form in a Coleosporium on asters, and it is doubtful if such be the case. In California, on the Monterey peninsula, Dr. E. P. Meinecke of this office found last spring a species of Cronartium on *Quercus agrifolia* Née in direct proximity with the galls of *Peridermium harknessii* occurring on the Monterey pines (*Pinus radiata* Don). No other gall-forming Peridermium was found on the peninsula. This Cronartium is morphologically identical with *Cronartium quercuum* (Brond.) Schr. For three successive years attempts have been made in the forest pathological greenhouse at Washington to successfully inoculate the leaves of eastern oaks with the aeciospores of *Peridermium harknessii*, without success. This, however, is negative evidence, and it may be that the spores used in the inoculations had lost their vitality during the period required for their shipment from the far west to Washington. No attempt has been made, to my knowledge, to artificially inoculate *Quercus agrifolia* with *Cronartium quercuum* in its eastern form. From this it appears that *Peridermium harknessii* Moore and *Peridermium cerebrum* Peck may be synonymous, since the type material of the former was collected on *Pinus radiata* in California in the Monterey peninsula. If such be the case, the other forms of *Peridermium harknessii* occurring on other species of pines in the west where no oaks are found, are either new species, or *Cronartium quercuum* in its western form must occur on some other hosts beside the oaks.

3. PERIDERMIUM MONTANUM Arth. & Kern

This year no extensive search was made for this species of Peridermium on the lodgepole pine (*Pinus contorta* Loud.). In 1911 it was collected in abundance in June on the lodgepole pine by Forest Ranger George, and later in the season by the writer, in the Gallatin National Forest, south of Bozeman, Montana. A search this year by Mr. George was made in the same localities. He was unable to find either the Peridermium on the lodgepole pines or the attendant Coleosporium found by the writer so abundantly on asters in proximity to the diseased trees. In other portions of the same forest this year, however, a species of Coleo-

sporium occurred sparsely on the leaves of wild asters and it is possible that the heavy infections of pines may occur infrequently.

4. PERIDERMIUM COLORADENSE (Dietel) Arth. & Kern

A number of national forests were visited in Colorado this year in which both *Picea engelmanni* Eng. and *Picea parryana* (André) Parry occur. The latter species grows as a rule at a lower altitude and was found more commonly attacked by *Peridermium coloradense* than the former. Many trees diseased by this fungus were noted, especially in the Rouett, Arapahoe, and San Isabel national forests in Colorado and in the Manti national forest, Utah. A large number of these were in a dying condition, which was due, in part at least, to the effects of the fungus.

5. MELAMPSORELLA ELATINA (Alb. & Schw.) Arth.

In the Rouett national forest this species of rust is common in its aecial form on *Abies lasiocarpa* (Hook.) Nutt., and its effect on the trees is decidedly injurious. In the Manti national forest the effect of the fungus is even more marked on this host.

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POWDERY SCAB OF POTATOES

Spongospora Subterranea (Wallr.) Johns.

H. T. Güssow

With Plate IV and One Text Figure

Some few months ago I received from various localities in Canada samples of potatoes affected with "powdery" or "corky" scab, a disease well-known in Europe, but as far as I was able to ascertain hitherto not reported as "established" on any part of the continent of North America. The first samples were sent by some French farmers in the Province of

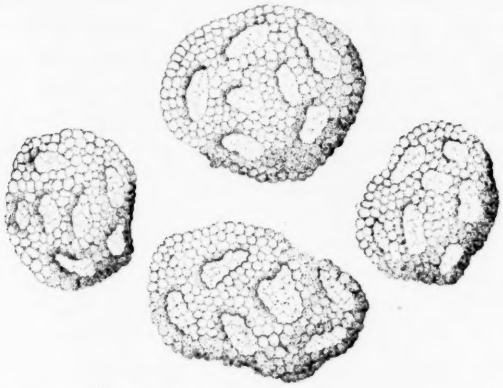

Fig. 1. Four spore "balls" of the fungus Spongospora. Apparently hollow perforated bodies composed of a large number of spores.

Quebec who had received a copy in French of the "Potato Canker Danger" poster issued by this Division in August, 1912. The French term for potato canker is "gale noire," which is the literal translation of the term "black scab" unfortunately chosen in England to describe potato canker. "Gale noire," however, is a term which describes the external appearance of the "powdery scab" very accurately, as may be judged from an examination of the accompanying plates, so that the action of the French farmers can be easily understood.

The plant pathologist will readily distinguish this form of scab from the Oospora, or common scab, by the characteristic spores of the fungus causing this trouble. Farmers, however, are liable to confuse it with the

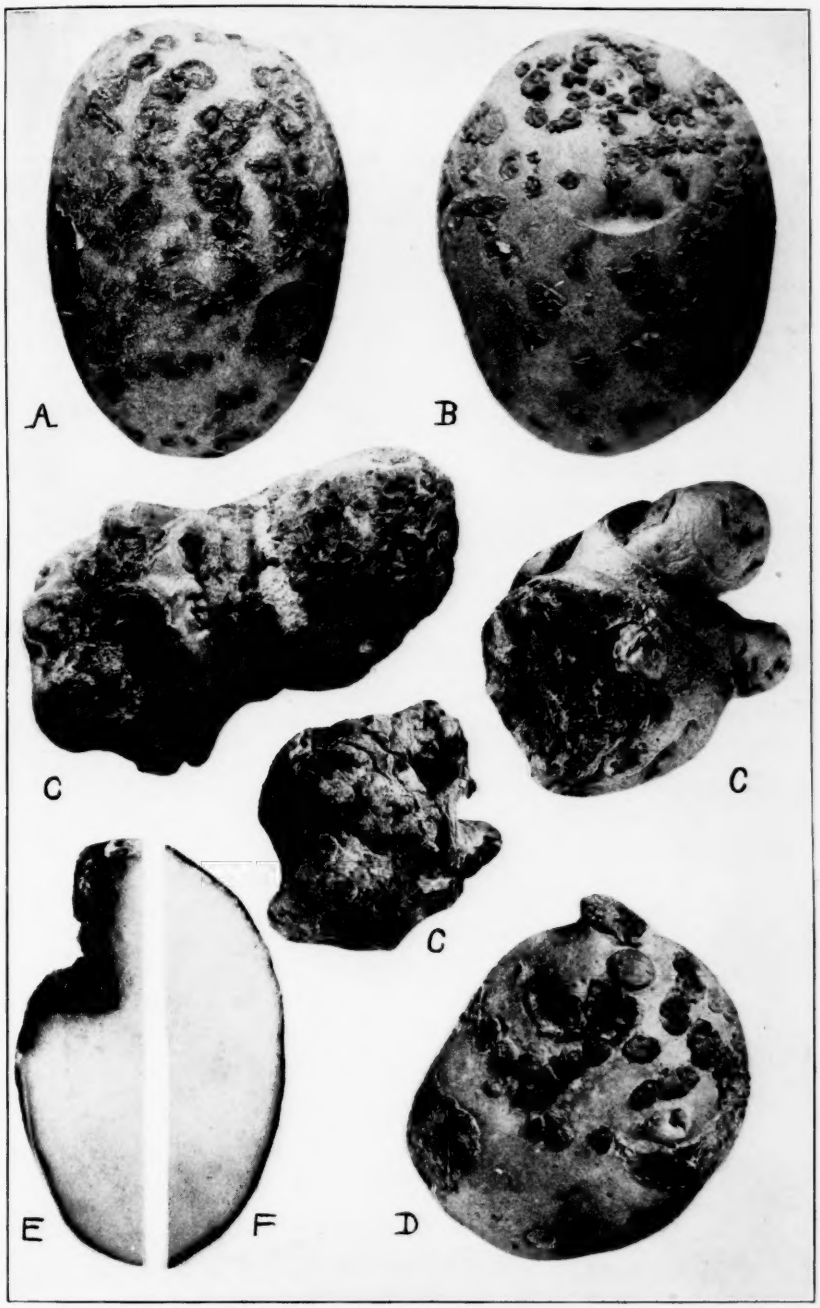

PLATE IV—SCAB OF POTATOES

a, Oospora scab. *b*, Spongospora scab; note close resemblance in this stage. *c*, Three tubers badly malformed by powdery scab. Sori are confluent; sometimes more than half of the tuber is destroyed. *d*, This tuber shows very characteristic Spongospora lesions, some still covered by the original thin membrane, which on bursting reveals the olive-brown powdery scab. *e*, Section through tuber affected by Spongospora scab. *f*, Section through tuber affected by Oospora scab.

ordinary scab, especially in the earlier stages. This was the experience in Canada, the farmers claiming to have known the "scab" since childhood—a period which in many cases appeared to date some forty or fifty years back. Since the first account of this disease in Europe by Brunchhorst appeared in 1886 it seems hardly probable that the disease was present so long ago in Canada.

Some of the more important literature on the subject is cited at the end of this note. Nothing new is to be added beyond the establishment of the first record, if I am correct, of its occurrence in North America. The disease seems well established in some counties of the Province of Quebec, while some quite isolated cases in widely separated regions in Canada (Cape Breton, Nova Scotia, New Brunswick, Ontario, Alberta) indicate the introduction of the disease by the use of affected seed potatoes. Recent specimens of potatoes received from Newfoundland also showed the presence of this disease in that country. These facts suggest that powdery scab probably occurs in the United States, also. It should be looked for especially in cases where seed potatoes have been imported from Europe.

The disease should by no means be regarded lightly. Severe attacks occur when potatoes are planted year after year on infected land. Where this is practiced the result will be potatoes hardly superior in quality to those badly affected by canker. This fact is worthy of notice, especially since, as in the case of canker, no preventive measures have proved of much value. It is my intention to establish a field laboratory in Quebec, in order to test probable means for controlling the disease.

Specimens of potatoes affected with powdery scab are at the disposal of any of my American colleagues, who will kindly intimate to me their desire of receiving the same.

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A ROT OF GRAPES CAUSED BY CRYPTOSPORELLA VITICOLA

C. T. Gregory

With Two Figures in the Text

While determining the amount of black rot in clusters of Niagara grapes during the latter part of September, 1911, a large number of berries were observed which were very recently rotted or only slightly spotted with the rot. Since this is not the usual action of the black rot fungus on ripe grapes, the writer was curious to see in what condition the fungus could be to cause such late infections. An examination of numerous cross sections of these mummied berries showed that they were not produced by the black rot fungus but by one having oblong spores borne in flattened pycnidial chambers.

The lesions on the naturally white berry are dark bluish-purple but become more brown as they grow larger. They occur at any place on the berry, but apparently center about the lenticels, spread slowly and finally involve the entire berry. When the spots cover about one-half of the fruit there appear within them small black bodies which gradually become more prominent, until in the mummy they show as relatively large projections thickly scattered over the surface. These are the pycnidia. Under moist conditions the spores ooze abundantly from them.

The differences between these mummies and the black rot mummies are: (1) they do not become so completely shriveled and hard as do the latter; (2) their color is a very dark blue as compared with the dead or brownish-black of the black rot; (3) the pycnidia are much larger and less numerous.

Numerous cultures were made from the mummies and also from beneath lesions of various sizes, thus eliminating the possibility that any fungus obtained could be a saprophyte following the black rot. In these cultures there soon developed an abundant white mycelial growth which eventually produced globose pycnidia of various sizes. There oozed from these pycnidia a yellow wormlike mass of spores. In appearance the cultures are exactly like those obtained from necrotic vines affected with the dead-arm disease. Examination of the spores show them to be of the same size and shape as those occurring in the mummies, i.e., oblong, and measuring 7–12 x 2–4μ.

Stained microtome sections of pycnidia from the cultures and from the berries were prepared. The pycnidia in the berry are relatively small

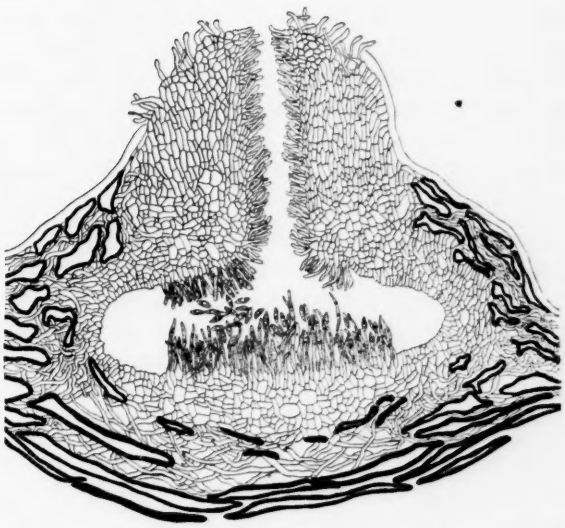

FIG. 1. A pycidium from a mummied grape. Such forms are most abundant in the grape, while in culture the multilocular forms are more abundant.

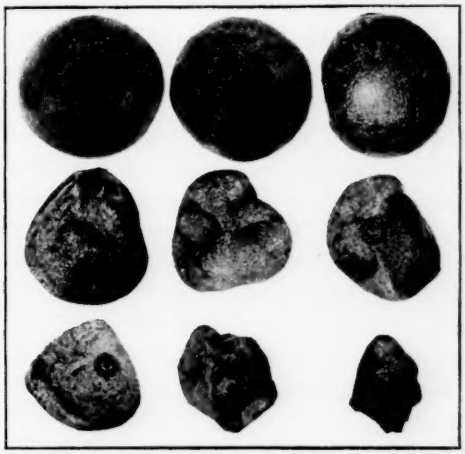

FIG. 2. Stages in the rotting of the grape. The first two stages showing discoloration centering about the lenticels.

and flattened, and the entire cavity is lined with basidial cells bearing the spores. Between these are other sterile threads which extend beyond the spores. The only difference between the pycnidia from the culture and from the berry is that the former are usually considerably larger. The cavity may be unilocular but it is ordinarily composed of several loculi. In the cultures there is often a large stroma containing numerous pycnidia.

When compared with cultures of *Cryptosporella viticola* in its conidial stage, it is seen that the spores are the same and, moreover, there are numerous cases where typical scolecospores were found in the cultures made from the berries. The fact that certain cultures do not produce these scolecospores does not indicate that the fungus is not *Cryptosporella viticola* (Red.) Shear, since numerous cultures of the type *Fusicoccum viticolum* Red. were examined and in some cases these organs were not found.

The mummies were left on the ground over winter in the hope that a perfect stage would develop, but in the spring only pycnospores were found. These readily germinated when placed in water and thus, in a way, obviate the necessity for the perfect form.

Inoculations were made with the spores, from pure cultures, on clusters of Niagara and Malaga grapes suspended in a moist chamber. In eighteen days the typical rot appeared and the berries were finally mummified. The same fungus was reisolated from these berries. Spores from pure culture, in sterile water, were sprayed on young grape vines in the greenhouse and kept moist for a few days. In about a month typical necrotic lesions appeared on the stems of certain of the vines. From these stems the fungus was again reisolated. When left in the moist chamber for some time there oozed from the lesions curls of spores which were the same in size and appearance as those obtained from the berries.

During the summer of 1912 numerous inoculations were made on grape shoots which were enclosed in lamp chimneys. About 75 per cent of the inoculations were successful. No attempt was made to reisolate the fungus. All attempts to infect ripe Delaware grapes were unsuccessful; no mature Niagaras were available at the time.

The rot was again found in 1912 at Romulus, N. Y., on the variety Niagara, and the same fungus was isolated.

CONCLUSIONS

The berries are attacked shortly before, and at maturity, by the same fungus that causes the dead-arm disease of the grape vine, producing a rot which in all its stages of development is very similar to black rot.

On close examination the two can be distinguished, but the most apparent difference is that this fungus attacks the grapes only when nearly ripe, while the black rot ceases to spread extensively in New York vineyards after the first of August.

Cultural characteristics, the characters of stained sections, the presence of scolecospores, the successful inoculations on the stem which produce typical necrotic lesions, together with the reisolation of the same fungus from artificially infected tissue, all combine to show that this disease is produced by *Cryptosporella viticola*.

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STUDIES ON THE FUSARIUM PROBLEM

H. W. Wollenweber

With Plate V and One Text Figure

A. THE DETERMINATION OF SOME ASCOMYCETES FROM THEIR CONIDIAL STAGE[1]

I. Unreliability of the Stroma as a Taxonomic Character

Before proceeding to the main discussion a few introductory remarks on the taxonomic value of the stroma will be useful. Fries (1849) has overvalued the presence and absence of a stroma in his whole system. He differentiates "stromate verticali" (e.g., Cordyceps), "stromate horizontali sessili" (e.g., Hypocrea, a subgenus of which is his Hypomyces "hymenio fungorum stromatis vices gerente"), "stromate nullo, sed saepe mycelio tuberculoso, crustoso. . . . . (e.g., Nectria). This system, somewhat modified, is still in use; see Lindau in Engler & Prantl's Natürliche Pflanzenfamilien, Seaver (1910) in North American Flora, etc.

[1] Definitions of the technical terms employed are as follows:

The *stroma* is the bed at the base of sporiferous stages and is always present. It may be reduced to scattered hyphae, within or on the host, or may form an aërial or immersed mycelial layer, the consistency of which is either loose or plectenchymatic (pseudoparenchymatic). The basal part of the stroma sometimes develops coremia (Claviceps, Sphaerostilbe).

A *sclerotium* consists of plectenchyma (pseudoparenchyma), either sterile, ball- or nodule-like, and functioning as a resting stage, like chlamydospores, or fertile, as the base of the stroma of perithecia and sporodochia.

A *chlamydospore*, in the true sense, is one- or two-cellular, formed terminally on the main hypha or its lateral branches, or intercalated within hyphae or conidia. It may also occur in chains or clusters, but must not be confused with the young stage of thallus-like plectenchyma, which can be produced in almost any species. The chlamydospore is characterized by a thick membrane consisting of more than one layer, and is long-lived (figs. *t*, *v*).

Pionnotes is merely a biological term for an effuse conidial stage, with a maximum of conidia and a minimum of aërial mycelium, which, as a rule, is slimy when young and resin-like or powdery-dry in old age. Its effuse masses may cover the naked surface of the host, reducing the stroma to endobiotic hyphae, or it may be embedded in a thallus-like plectenchymatic or hyphasmatic stroma.

Sporodochium. The chief contrast to pionnotes lies in the Tubercularia-like hemispherical form of the sporodochia, which are found scattered individually and in colonies. Their stroma may or may not have a plectenchymatic base (sclerotium).

Doubts, however, as to the value of the basis of Fries' system have been frequently expressed. These doubts have been recently confirmed by careful comparative studies of exsiccati (v. Höhnel, 1909, Weese, 1911) and by pure culture studies of ascomycetes and fungi imperfecti (Brefeld). Studies on Neocosmospora led Erw. F. Smith also to the conclusion that the stroma is one of the most variable factors, while Appel and Wollenweber showed that Fusaria, with and without an ascus stage, behave similarly.

The stroma in itself was especially changeable in pure cultures of Gibberella, Melanospora, Hypocrea, Mycosphaerella, and Calonectria. It may, however, be of taxonomic value in extreme cases, when it entirely encloses the perithecia or grows with a coremium-like base (Claviceps, Cordiceps, Xylaria). Intermediate groups, such as the Hypocreaceae, having free perithecia, or with the latter partly covered by the stroma, are often hardly to be distinguished from Nectrieae, since on certain substrata in pure culture their perithecia grow entirely superficially, while on others they are slightly immersed in the stroma. As the same fact is true within genera of Nectrieae, including Nectria itself, it will be difficult or almost impossible to follow Seaver's proposition to divide the Hypocreales into groups according to the presence or absence of the stroma.

II. PURE CULTURE METHOD FOR DIFFERENTIATION OF SPECIES

The results from pure grown fungi are encouraging in so far as they seem to be the first step to a better understanding of the relationship of families and lower groups of the ascomycetes. Both the ascigerous and conidial stages of the Hypocreales offer excellent material for research work of this kind. The conidial stage, however, has been entirely neglected by many authors. The reason lay in the lack of a culture method answering all purposes. We need, therefore, a pure culture method which gives the normal stages and, if possible, all stages, of a fungus.

From my experience, many of the fungi in question grow normally on steamed stems of trees, shrubs, and herbaceous plants. Conidia, chlamydospores, and perithecia, which are sufficiently characteristic to decide the determination, develop on these substrata. Secondary characters are often obtained from cultures on rice, potato tubers, and other starchy media, upon which some fungi show brilliant colors, extra large sclerotia and stromata. Some of these characters may be reduced or absent on stems. The same condition was found in nature, as evidenced by the discovery on dry wheat culms of perithecia of *Gibberella Saubinetii*, unaccompanied by either stromata or sclerotia, while some soft stalks of *Zea mays* bore perithecia on large sclerotia, and on a seed ball of potato an

effuse plectenchymatic stroma developed. Under normal and constant conditions pure cultures of this Gibberella gave the same modifications of mycelial growth, but almost constant fruiting stages.

The degree of maturity of vegetable tissue has a bearing on the development of the complete life cycle of some wound parasitic fungi: e.g., *Calonectria graminicola* (Berk. & Brm.) Wr., a cause of seedling blight and snow mould disease of cereals, produced perithecia more freely on green parts which were cut at the time of flowering than on parts cut at maturity. The fact that this fungus is cultivable on immature tissue is another indication of its special adaptation to the seedling stage of the host.

Twenty ascomycetes belonging to widely different groups, especially of Hypocreales and Sphaeriales, were proved to be cultivable. They permitted a comparative study of conidial stages of various genera. As a result of these comparisons it can be stated, that within some genera, such as Gibberella and Melanospora, all species have conidia of uniform shape, while different species may differ in the size, septation, and color of these organs. Nectria, however, has several sections, one with cylindrical, another with sickle-shaped and pointed septate conidia, a third with small unicellular ellipsoidal conidia.

III. Division of Fusaria into Sections

The whole genus Fusarium (Link) may be divided into sections in order to unite species having related characters, the most important of which is a uniform shape of conidia. Some of these resemble the conidial stage of ascomycetes, e.g., Martiella, which resembles subnormal conidia of Hypomyces (Fig. D).

We are still far from having a conclusive proof, however, of the widely recognized theory that Fusarium is the obligate conidial stage of ascomycetes. Such Ramularia- or Septocylindrium-like stages as have conidia of cylindrical shape but no chlamydospores in the true sense, often develop an ascus stage (Mycosphaerella, Nectria) in pure culture. Other fungi differ, however, in the absence of an ascus stage and the presence of chlamydospores. Such species are, therefore, true Ramularia (fig. R) or Septocylindria, and are not to be confused with the ascomycetes on account of the resemblance of a particular character, such as conidial form. The same is true in *Fusarium rubiginosum* (fig. G), *F. discolor* var. *sulphureum* (fig. H), and *F. trichothecioides* (fig. J), which developed intercalated clusters and chains of chlamydospores but no perithecia, while *Gibberella Saubinetii* (fig. K) had perithecia but no chlamydospores. This difference readily distinguishes *F. rubiginosum* from Gibberella, and the discovery is of taxonomic importance because the

conidia of the two fungi, when cultivated separately in a number of strains isolated from various hosts, agree sometimes more closely than appears in the illustration. Hence the above results furnish a good means for determination in that the *presence of chlamydospores within Ramularia, Septocylindrium and the Fusarium section Discolor indicates the absence of perithecia.* However, the fungus Hypomyces (fig. D) has a conidial stage in young cultures similar to the Fusarium section Martiella (figs. A–C). It also has chlamydospores as shown in figures T and V. Thus Martiella and the conidial stage of Hypomyces are determinable only by means of the more subtle morphological details of the conidia.

This exception seems to diminish the value of the chlamydospore in the determination of ascomycetes from their conidial stage. Furthermore, some Nectria species, e.g., *N. vandae* and *N. Goroshankiniana* Wahrl., are said to have chlamydospores in their conidial stage. I think it better to transfer such fungi to Hypomyces in order to get as distinct a border line between the two genera as possible. This proposition is more than an hypothesis, being the result of comparative studies on many ascomycetes grown in pure culture for a long time without a loss of vital power. Other factors, such as stromata, conidia, ascospores, and perithecia are often constant under uniform conditions, but variable on different substrata. They represent good characters for sections and lower groups, but are of doubtful value for the generic border line between Nectria and Hypomyces.

A careful study of exsiccatae of Hypomyces leads René Maire (1911) to separate species with obtuse ascospores from Plowright's (1882) Hypomyces and to establish for them the genus Nectriopsis. According to him a Hypomyces ascospore ought to have an apiculus at each end. In pure culture, however, I find apiculi, or sharply pointed ends, only on young ascospores, with more or less obtuse ends in maturity. This fact proves how difficult it is to base taxonomic suggestions merely on studies of exsiccatae, when we have no criterion of the degree of maturity of the organs investigated.

If we state that, in contrast to Nectria, Hypomyces has true chlamydospores, we have an excellent differential character between both genera. Then *Nectria ipomoeae* Halsted (1891) is a true Hypomyces.

B. TAXONOMY AND PATHOLOGY OF THE FUNGI

Proceeding from the supposition that the diagnosis of parasitic fungi should be based on conclusive proof of their pathogenicity, the last year has been principally spent in making infection experiments. Thus the diagnoses given in the following part represent, if not otherwise stated,

both the taxonomic and the pathological results obtained by the author and referred to in the last parts of this paper: "The wilt disease problem" and "Tuber rot and ring discoloration of the potato."

I. FUSARIUM (LINK)

The sections Martiella (figs. A–C), Elegans (E, F, S), Discolor (G–J), Gibbosum (L, M), Roseum (N), and Ventricosum (Q) are provisionally established here in order to facilitate the arrangement of the species.

a. SECTION ELEGANS

All species have scattered, ellipsoidal, unicellular conidia, averaging 5–12 x 2–3.5μ in size. Many species have also sporodochia with a sclerotial base and pionnotes. The sickle-shaped conidia have the form of the Elegans type (figs. E, F, S), mostly 3-septate, but also 4- and 5-septate. The average size differs according to the species, the majority having 3-septate conidia, 25–40 x 3–4.5μ, while the 4-septate are somewhat larger, and the 5-septate average 40–50 x 3–4.5μ. Conidia in masses, mostly salmon-colored, but in some species brownish white, in others brilliant orange. Conidiophores verticillately branched in sporodochia (fig. U). Chlamydospores ellipsoidal, terminal and intercalated (fig. T, V), the unicellular ones being 5–10μ, the 1-septate ones up to 12μ. Ascigerous stage unknown.

Species in this section are: *Fusarium oxysporum* (Schlecht.), *F. tracheiphilum* Smith, *F. vasinfectum* (Atk.), *F. lycopersici* Sacc., *F. niveum* Smith, *F. redolens* n. sp., *F. orthoceras* App. & Wr., *F. conglutinans* n. sp., notes on which follow.

1. *Fusarium oxysporum* (Schlecht.) (Fig. F).

Sporodochia and a reduced pionnotes present, in masses salmon-colored, the conidia of both being 3-septate up to 100 per cent, 25–45 x 3.25–4.5μ, 4-septate up to 25 per cent, 5-septate up to 10 per cent, where they are 40–50 x 3.5–4.75μ in size. Sclerotia blue on steamed potatoes. A slight lilac odor on steamed rice, milk, etc. Vascular parasite, cause of wilt disease, but not tuber rot, of *Solanum tuberosum* in the United States of America, possibly also in southern Europe and the potato districts of South Africa, Australia, etc. Also found on various hosts, such as *Solanum lycopersicum*, Vigna, Pisum and Ipomoea. The disease associated with this fungus has been confused with "leaf roll," a potato disease, called "Blattrollkrankheit" in Germany.

2. *Fusarium tracheiphilum* Smith

Closely related to *F. oxysporum* (Schlecht.), but without pionnotes, and odorless. Vascular parasite, cause of wilt disease of Vigna in the United States of America. A foot disease of the same host has also resulted from greenhouse inoculation. Saprophytic on various hosts.

3. *Fusarium vasinfectum* (Atk.)

Closely related to *F. oxysporum* (Schlecht.), but with a perfect pionnotes and a very strong lilac odor on steamed rice, milk, etc. Vascular parasite, cause of wilt disease of *Gossypium herbaceum* and *G. barbadense* in the United States of America, probably also in the cotton growing districts of other continents. Saprophytic on various hosts.

4. *Fusarium vasinfectum* (Atk.), var. **inodoratum** n. v.

Differs from *F. vasinfectum* (Atk.) by the absence of odor. Vascular parasite, cause of wilt disease of *Gossypium herbaceum* and *G. barbadense*. The same geographical distribution as *F. vasinfectum*.

5. *Fusarium lycopersici* Sacc.[2]

Differs from *F. oxysporum* (Schlecht.) in having conidia of a little larger average size, a perfect pionnotes, colorless sclerotia on steamed potatoes, and no odor. Vascular parasite, cause of wilt disease of *Solanum lycopersicum* in all parts of the United States of America, except the most northern: probably also in southern Europe. This fungus never causes such a progressive rot of the fruit as *F. sclerotium* n. sp. and *F. falcatum* App. & Wr., both of which are wound parasites of the tomato.

6. *Fusarium niveum* Smith

Differs from *F. oxysporum* (Schlecht.) in having larger conidia and no odor; from *F. lycopersici* in having blue, somewhat larger, sclerotia on steamed potatoes. Vascular parasite, cause of wilt disease of Citrullus in the United States of America.

7. **Fusarium redolens** n. sp. (Fig. E)

Differs from all the above-mentioned species in the large size of its triseptate conidia, 30–40 x 4.5–5.5μ, and in the color of the brownish-white conidial masses. A lilac odor is produced on rice and milk. No

[2] Under *F. oxysporum* Schlecht. in Sacc. Syll. Fung. **4**: 296.

blue sclerotia. Vascular parasite, cause of wilt and foot disease of *Pisum sativum*. Distribution unknown.

8. *Fusarium orthoceras* App. & Wr. (Fig. S)

Conidia less pointed at the apical end than typical species of the section Elegans, the base very reduced, pedicellate or papilla-like. No sporodochia, no pionnotes and no sclerotia. Triseptate conidia very rare, 25–40 x 3.25–4μ, never over 15 per cent being 3-septate. A cosmopolitan species. Inhabits the root system and the tubers of Solanaceae. Probable cause of jelly-end rot of the potato tubers. Also found on various hosts and isolated from the soil. The confusion of this species with *F. oxysporum* by Appel & Wollenweber (1910, p. 146) arose from their study of an American culture sent as "probably *F. oxysporum*" which is now known to have been *F. orthoceras*.

9. **Fusarium conglutinans** n. sp.

Differs from *F. orthoceras* in the absence of a wine-red color on rice, which is a striking character of typical species of the section Elegans. Vascular parasite, cause of wilt disease of *Brassica oleracea* var. *capitata* (proved by Erwin F. Smith, L. R. Jones, L. L. Harter) in the United States of America.

OTHER FUSARIUM SECTIONS CONTAINING WOUND PARASITES

Other sections, such as Martiella, Discolor, Gibbosum, Roseum, and Ventricosum, have also been proved parasitic and show a striking contrast to the section Elegans in being able to destroy parenchymatic tissue, while Elegans contains purely vascular parasites only.

b. SECTION MARTIELLA

Contains species having conidia of the form-type shown in Figures A, B, C. Chlamydospores present. No known ascus stage.

1. *Fusarium solani*(Mart.) (Fig. A)

Cf. Appel & Wollenweber, Grund lagen . . . . 1910.
Saprophyte common on potatoes and various other hosts both in Europe and America.

2. *Fusarium Martii* App. & Wr. (Fig. B)

Cf. Appel & Wollenweber, Grundlagen . . . . 1910.
Saprophyte having the distribution of *F. solani* (Mart.).

3. *Fusarium coeruleum* (Lib.) (Fig. C)

Cf. Appel & Wollenweber, Grundlagen . . . . 1910.
Wound parasite, cause of dry rot of potato tubers both in Europe and America.

c. SECTION DISCOLOR

Conidia of the form type shown in figures G, H, J. Intercalated chlamydospores. Ascigerous stage unknown.

1. *Fusarium rubiginosum.* App. & Wr. (Fig. G)

Cf. Appel & Wollenweber, Grundlagen . . . . 1910.
Differs from *Gibberella Saubinetii* (Mont.) Sacc. mainly in the presence of clusters and chains of intercalated chlamydospores and the absence of an ascus stage. Wound parasite on cereals, cause of scab and seedling blight (foot disease) in Europe and the United States of America (proved by unpublished studies of E. C. Johnson, Washington, 1912), also found on potato tubers, where it is, however, less active than *F. coeruleum*, *F. discolor* var. *sulphureum*, and *F. trichothecioides*. Saprophyte on various other hosts.

2. *Fusarium discolor var. sulphureum* Schlecht. (s. sp.) (Fig. H)

Cf. Appel & Wollenweber, Grundlagen . . . . 1910.
Wound-parasite, cause of dry rot of potato tubers in Germany.

3. *Fusarium trichothecioides* Wr. (Fig. J)

Cf. Jamieson & Wollenweber . . . . 1912.
Wound parasite, cause of dry rot of potato tubers in the United States of America.

d. SECTION GIBBOSUM

Conidia of the form-type figured in figure L, M. Intercalated chlamydospores. Ascigerous stage unknown.

1. *Fusarium falcatum* App. & Wr. (Fig. L)

Cf. Appel and Wollenweber, Grundlagen . . . . 1910.
Conidia 5-septate, 35–55 x 4–5μ, parabolically curved. Wound parasite, cause of tomata fruit rot in Germany and the United States of America.

2. *Fusarium gibbosum* App. & Wr. (Fig. M)

Cf. Appel and Wollenweber, Grundlagen . . . . 1910.

Conidia 5 (5–7)–septate, 30–60 x 4–5.25μ, hyperbolically curved. Probably also a cause of potato tuber rot, but the results of inoculation experiments were not as uniformly positive as in the case of the other wound parasites mentioned.

3. **Fusarium sclerotium** n. sp.

Differs from *F. gibbosum* App. & Wr. (fig. M) in having spherical, blue sclerotia. Conidia mostly 5-septate. A wound parasite like *F. falcatum* App. & Wr. (fig. L) the cause of fruit rot on *Solanum lycopersicum* and *Citrullus vulgaris* in the United States of America.

e. SECTION ROSEUM

Conidia of the form-type shown in figure N. No true chlamydospores, but as a rule sclerotia, blue colored in a number of species. Ascigerous stage unknown. The majority are to be regarded as wound parasites, some causing scab and seedling blight, others fruit rot.

1. *Fusarium subulatum* App. & Wr. (Fig. N)

Cf. Appel & Wollenweber, Grundlagen. . . . . 1910.

A cosmopolitan species, saprophytic on almost any dead substance, also found in water, and in the soil, wound parasitic under conditions of high humidity and closed air only, cause of scab and seedling blight on cereals; of dry rot on potato tubers, but less active than *F. coeruleum* (Lib.) and other Fusaria.

f. SECTION VENTRICOSUM

Conidia of the form-type shown in figure Q. No sporodochia. Chlamydospores similar to those figured in figure T and V. Ascigerous stage unknown.

1. *Fusarium ventricosum* App. & Wr. (Fig. Q)

Conidia never formed in sporodochia, brownish-white to cream-colored, 3-septate, 29–37 x 5.75–7.5μ. Conidiophores bostryx-like or irregularly branched. Chlamydospores like those of the section Elegans. Wound parasite, cause of a tuber rot of *Solanum tuberosum* in the United States of America, also found in Europe. Inhabits also *Beta vulgaris*.

II. VERTICILLIUM

Verticillium is a genus having no morphological relationship to Fusarium but causing similar pathological symptoms.

a. *Verticillium albo-atrum* Reink. & Berth. (1879)

No sporodochia, but black sclerotia. Conidia as a rule unicellular, ellipsoidal, 5–12 x 2–3.5μ. Conidiophores verticillately branched. Vascular parasite, cause of wilt disease on *Solanum tuberosum*, *S. melongena* and *Hibiscus esculentus*[3] in the United States of America, also in Germany on *Solanum tuberosum*, cause of the Kräuselkrankheit described by Reinke & Berthold, a typical wilt disease which should not be confused with Blattrollkrankheit (leaf roll), the latter being probably a physiological trouble.

III. RAMULARIA

Unger (1833) Synonymy. *Septocylindrium* Bononden (1851). Ramularia is a genus related to Fusarium but with conidia of cylindrical shape. Chlamydospores are present, but the ascigerous stage is unknown. It should not be confused with the ascomycete Mycosphaerella, which has similar conidia but no true chlamydospores. Ramularia contains wound parasites causing root troubles, stem anthracnoses, leaf spot diseases and fruit rots.

1. **Ramularia didymum** (Hart.) Wr. n. n. (Fig. R)

Synonymy: *Fusisporium didymum* Harting (1846). *Fusarium didymum* (Hart.) Lindau (1909); Appel & Wollenweber (1910). This species is given here as an example in order to show the conidial type of the genus. A complete diagnosis will be published later. Conidia 1-septate, 20–30 x 4.25–5.5μ, also 2- and 3-septate, averaging up to 36 x 6.25μ in size. Inhabits the soil. Also found on Solanum and Rubus in connection with decay of the root system.

IV. ASCOMYCETES WITH FUSARIUM-LIKE CONIDIAL STAGE

The following ascomycetes should not be confused with true Fusaria on account of the resemblance of a particular character, such as conidial form: Neocosmospora, Hypomyces, Calonectria, Gibberella and Nectria.

[3] F. L. Stevens and G. W. Wilson (1912) in their study of okra wilt disease found black sclerotia in pure cultures of the parasite, which they call *F. vasinfectum*. To judge from this and my positive results obtained with Verticillium on okra, they may have worked with *Verticillium albo-atrum* and not with Fusarium.

1. *Neocosmospora vasinfecta* Smith (1899). (Fig. P)

Red ovoid perithecia 250–400 x 200–350μ. Ascospores unicellular, 10–12μ, if globose; 11–14 x 8–12μ, if ellipsoidal. Conidia ellipsoidal, mostly unicellular, 8–12 x 2.5–4μ, occasionally septate, the 3-septate conidia having an average size up to 32 x 4.75μ are neither pointed at the ends nor pedicellate at the base. Chlamydospores are absent. Saprophyte, common on decayed legumes and other plants in America, Asia and Africa. This fungus is often confused with wilt-disease fungi belonging to the section Elegans of the genus Fusarium.

2. **Hypomyces ipomoeae** (Hals.) Wr. n. n. (Fig. D)

Synonymy: *Nectria ipomoeae* Halsted (1891); *Creonectria ipomoeae* (Hals.) Seaver. (1910); *Nectria* (*Dialonectria*) *coffeicola* Zimmermann (1901); ? *Nectria saccharina* Berk. & Cook (Cf. Sacc. 1883, Syll. **2**: 489); ? *Nectria Goroschankiniana* Wahrlich (Bot. Zeit. **44**. 1886).

Red rugose ovoid perithecia 225–375 x 175–300μ. Ascospores 1-septate 10–13 x 4.5–6μ. Conidia similar to the section Martiella of the genus Fusarium, but more pronouncedly pedicellate at the base and more pointed at the apical end, 5-septate, 45–70 x 4.25–5.5μ, 3–5-septate, 30–70 x 3.75–5.5μ; if unicellular, 6–12 x 3-4.75μ. Chlamydospores 7–10μ. Saprophyte on decayed egg plants and sweet potatoes, United States of North America.

3. **Calonectria graminicola** (Berk. & Brm.) Wr. n. n. (Fig. W)

Synonymy:? *Nectria graminicola* Berkeley and Broome (Ann. Mag. Nat. Hist. 3 ser. **3**: 376. 1859); *Fusarium nivale* autorum pro parte; *Fusarium hibernans* Lindau (1909), *Fusarium minimum* Fuckel (Symb. p. 370. 1869).

Brown perithecia, average diameter 125–200μ (limits 75–300μ). Ascospores fusoid, 1–3-septate, 12–15 x 2.75–3.75μ. Conidia, like the mycelium, ochreous to salmon-colored, 3-septate, 23–26 x 3.25–4μ, formed as a slightly curved comma. Instead of true chlamydospores a plectenchyma is present.

Cause of seedling blight (snow mould disease) on cereals (first proved by Ihssen (1910)). Widely distributed both in Europe and North America.

4. *Gibberella Saubinetii* (Mont.) Sacc. (Fig. K)

Synonymy: *Gibberella cyanogena* (Desm.) Sacc. 1883; *Gibberella tritici* P. Hennings. (Hedwigia, 1902); *Gibberella tropicalis* Rehm. (Hedwigia, 1898).

Blue perithecia 150–250 x 100–250μ. Ascospores sickle-shaped, 3-septate, 20–30 x 3.75–4.25μ. Conidia similar to the section Discolor of the genus Fusarium, 3–5 septate, 30–60 x 4.25–5.5μ, but no true chlamydospores are present, while the section Discolor has clusters and chains of chlamydospores. Cause of kernel scab and seedling blight (foot disease) on cereals, (proved by Selby and Manns, 1909), widely distributed both in Europe and America on various hosts.

5. *Nectria galligena* Bresadola. (Fig. O)

Cf. Weese, Zur Kenntnis des Erregers der Krebskrankheit an den Obst.- und Laubholzbäumen. Zeitschr. f. d. landwirtsch. Versuchswesen in Österreich. 1911: 872–885.

Synonymy: *Fusarium Willkommii* Lindau (1909).

Red ovoid perithecia, 400–450 x 275–325μ. Ovoid ascospores 1-septate, 14–16 x 5–7.25μ. Cylindrical 5-septate conidia 57–73 x 4.75–6μ, but 30 % of all conidia may be 6-septate, average length up to 82μ. No true chlamydospores are present. Wound parasite, cause of canker on apple trees in Europe (proved by Aderhold, 1903, and confirmed by the author). Associated with canker on various other trees. This fungus is often confused with *Nectria ditissima* Tul., a probable saprophyte, which has shorter ascospores, 12–14μ long.

C. THE WILT DISEASE PROBLEM

Wilt diseases are very common and very destructive to various crops in this country. Some types of wilt are caused by bacteria; most of them, however, by fungi, in some cases by Verticillia, but especially by Fusaria. Thus the wilt disease problem is principally a Fusarium problem.

As far as we know, wilt occurs only in a few scattered families of monocotyledons. I recall Liliaceae (e.g., Allium, Asparagus), also Bromeliaceae (Ananas) and last, but not least, Musaceae (Musa, "Panama-disease"). Many families of dicotyledons, however, are seriously attacked.

Some series, such as the Tubiflorae, contain more than one family susceptible to wilt, viz., Solanaceae (Solanum, Capsicum) and Convolvulaceae (Ipomoea). Other series have but one family suffering from wilt disease, such as Leguminosae (Lupinus, Medicago, Indigofera, Cicer, Vicia, Pisum, Phaseolus, Vigna or cowpea, Cajanus), Malvaceae (Gossypium, Hibiscus), Linaceae (Linum) and Cucurbitaceae (Citrullus). Even the Cruciferae (Brassica), Compositae (Aster), Araliaceae (Panax), Caryophyllaceae (Dianthus) and Pedaliaceae (Sesamum) are not free from the disease. Such plants as Citrus have the same disease, but we do not know whether it is caused by a fungus.

The opening up of the Verticillium problem we owe to Reinke and Berthold (1879), that of the whole Fusarium problem to Erwin F. Smith (1899). The latter investigator obtained the first conclusive results in the etiology of the watermelon wilt by securing five hundred successful infections by simply inoculating the soil with pure cultures of the vascular parasite. He considered the organism a variety of the same species as causes cotton and cowpea wilt. The parasite of watermelon wilt was not infectious to cotton or cowpea, nor to tomato or potato, through soil inoculations.

Dr. Smith also discovered a very peculiar fact. A perithecial stage (Neocosmospora) was frequently associated with the parasite on cowpea, especially on parts already killed. In pure culture, derived from a single ascospore, all stages (mycelium, conidia, ascospores) of Neocosmospora redeveloped the ascus stage, and the conidia in subcultures of the fungus were similar to the small conidia of the parasite. Cowpea inoculations with the fungus derived from ascospores of Neocosmospora failed, however. Since this failure might be due to the fact that the natural method of infection had not been discovered, the author did not feel justified in regarding it as proof of saprophytism. Therefore, he chose the hypothesis that the vascular parasite and the ascomycete Neocosmospora were identical.

This hypothesis, although recognized for ten years, has been doubted recently by E. J. Butler, Pusa, India (1910), and by B. B. Higgins, Raleigh, North Carolina (1911). Both investigators consider Neocosmospora to be saprophytic. Butler did not get conclusive cases of wilt when he inoculated pigeon-pea, cotton, indigo or gram with subcultures from ascospores. Higgins found important differential characters in pure culture between the ascigerous and the conidial fungus.

After having worked for four years on the taxonomy of Fusaria and the study of their pathogenicity, I find it very easy to produce cotton, tomato, and cowpea wilt with the young pionnotes and sporodochial stage of the 3-septate conidia which the vascular parasites freely form in pure culture on steamed stems and potato cylinders. Other types of wilt, e.g., pea wilt, require more accuracy to eliminate contaminations. Details of the methods used will be given in a special bulletin.

Hypocotyl inoculations gave me the largest number of cotton and tomato wilt infections, often ten times as many as soil infections. A very slow partial wilt, often remaining latent for a long while, resulted from stem inoculation above ground. The whole plant frequently died from hypocotyl inoculations, although very slowly, especially in the case of full grown plants. All plants, however, which wilted from soil inocula-

tion, did so suddenly, dying from starvation after the root system became infected.

My results, then, are as follows: Subcultures from all known stages of the vascular parasite of one host, (i.e., mycelium, unicellular and 3-septate conidia, less frequently 4-septate and rarely 5-septate conidia, and chlamydospores) were found virulent if grown under normal conditions. By normal conditions, I mean those conditions which insure the greatest longevity for the fungus under consideration. In general, with fungi having a sporodochial stage, conditions which favor the production of this stage also favor its longevity and so are considered as most nearly normal.

None of the corresponding stages present in Neocosmospora gave a single positive case of wilt under similar conditions. It may be stated that Neocosmospora has no sporodochial or pionnotes stage and 3-septate conidia very seldom occur, always less than 1 per cent of the total number. The conidia are ellipsoidal, rounded at both ends, and never constricted at the apical end or pedicellate at the base (fig. P). The normal color is brownish-white. Chlamydospores are lacking.

The vascular parasite in wilt diseases, however, forms 3-septate conidia (more seldom 4- and 5-septate) in sporodochia and often in pionnotes stage, up to 100 per cent. They are constricted at the apical end and pedicellate at the base (figs. E, F). Chlamydospores are present (figs. T, V). In the majority of species the color of conidial masses is ochreous to salmon in the light. Species having these characters are placed in the section Elegans.

The wilt disease parasites differ from each other. One is distinguished by the absence of sporodochia and sclerotia (cabbage wilt), another has blue sclerotia (cowpea wilt), and a third has colorless sclerotia (tomato wilt). Some of them produce a pionnotes stage very freely (cotton and tomato wilt). This is entirely wanting in the cowpea wilt and scant in the potato wilt. Aromatic odors occur in cotton wilt and a few others. The average diameter of conidia and the color of conidial masses have also been found variable within the section Elegans. Biological characters are of secondary importance.

The differences of wilt disease parasites confirm the fact that we have to do with different species and varieties. The parasite from one host, as a rule, has not been found on living organs of another host. In pure culture the parasite from one host could not be transformed into the parasite of any other host and did not cause wilt in any other host as a result of inoculation experiments. Thus, at least ten wilt parasites can easily be distinguished by means of pure cultures. Some of these species have a close relationship to each other, but phylogenetic suggestions concerning

the range of evolution can not yet be given, for such can be based only on complete diagnosis of the parasite of each different wilt type.

Some of the most widely different wilt fungi are *Fusarium lycopersici* Sacc. *F. vasinfectum* Atk., *F. conglutinans* n. sp. (cabbage wilt), *F. udum* Butler[4] (pigeon-pea wilt), and *F. oxysporum* (Schlecht.). *F. niveum* Smith and *F. tracheiphilum* Smith are distinct from each other, both of them being more closely related to *F. oxysporum* than to any others. The Fusarium of bean wilt and *F. cubense* Smith (banana wilt) resemble *F. vasinfectum.*

A particular wilt disease of a certain host may be caused by more than one strain of the same species. This fact has been proved for cotton wilt. One of the parasitic cotton wilt strains produced in culture a very strong aromatic odor which fluctuated between turpentine and lilac and was probably caused by more than one aromatic substance. This odor was entirely absent in another strain and its subcultures. More than one species, differing both in size of conidia and color of conidial masses, may cause the St. John's disease of the garden pea.

Since all the experiments in the greenhouse and in nature have been carried out on a limited number of plants (never more than one hundred) the positive results should be regarded only as a first step, in order to give a general view and to outline the problem. They confirm, however, Butler's and Higgins' doubts of a genetic connection between Neocosmospora and the vascular wilt parasites, and show that Neocosmospora is an obligate saprophyte. They prove that a series of wilt diseases are caused by species differing both in morphology and biology, but belonging to the same section of the genus Fusarium. Further studies on the differentiation of wilt fungi should furnish also a basis for the arrangement of crop rotations, in order to prevent infection of one crop by the parasites of the preceding crop.

A method of controlling wilt diseases which is richer with prospects seems to be promised by the selection of resistant plants. This was first successfully performed by W. A. Orton (1900–1908) with cotton, watermelon and cowpea. An advantage of this method lies in the fact that it is independent of the question whether the disease is due to microorganisms (bacteria, fungi), on the one hand, or to external conditions (lack of water, hot weather, etc.), on the other. By selection one secures adaptation to the chief defects of the environment. Rotation as a pre-

[4] I propose the name *F. Butleri* for this species, because the name *F. udum* has already been used by Berkeley (1841) and refers to a distinct old species, well known by its pionnotes stage covering the cut surface of oak, elm and other trees. It is also found on Irish potatoes, tulip bulbs and in the soil. It was temporarily transferred by Saccardo (1886) to the genus Pionnotes, which, however, has no sound morphological basis (Appel & Wollenweber, Grundlagen . . . . 1910).

ventive measure is also partly independent of etiological questions in so far as it regulates such external conditions as the food supply. If, however, the length of the rotation is to be determined, it is necessary to know whether or not a microörganism is the cause, since a fungus may remain living several years in the absence of its special host.

A knowledge of the identity of the fungus is very important, especially with the wilts, since they are mostly caused by Fusaria, and this genus bears conidia of such similar appearance that wilts in different genera of host plants may be produced by what look like the same Fusarium species, e.g., *F. vasinfectum*. If this were so, one might expect the same species to infect all plants susceptible to wilt, or, if the fungus was a highly adapted parasite, that it would mainly attack one host, but would possibly adapt itself gradually to others. Whether such adaptation occurs and causes changes in the nature of the parasite, indicated in pure culture by differences in general appearance, production of color, etc., has not been determined.

While science alone gropes in darkness, its union with practice has succeeded in preventing the wilt from getting the upper hand. By the selection of resistant strains cotton, cowpeas and watermelons are now successfully cultivated on badly-infected soil. On the other hand, by attention to crop rotations practice itself has, independent of science, worked successfully on the control problem. It has been found from long experience that cotton and cereals grow well after potatoes, after which, of course, other vegetables requiring intercultivation, such as beets cabbages and turnips, may follow. For different climatic conditions different crops are used in rotation. Successful practice has shown that the more widely different the hosts, the shorter the period of rotation required. This, in the light of the Fusarium wilt problem, seems to confirm the fact that parasites differ proportionally to the taxonomic differences between the respective hosts, and the possibility of the adaptation of the parasite decreases proportionally to the taxonomic distance of the host. In contrast with this hypothesis stands the fact that the banana wilt fungus bears, so far as we know, the closest relationship, or is perhaps identical, with the cotton wilt fungus. Furthermore, the Fusaria of the tomato and Irish potato wilts seem to differ so widely that one may easily be inclined to recommend the growing of these crops in continuous rotation, unless other factors stand in the way, such as one-sided exhaustion from related Solanum species, or different climatic adaptations. It is desirable, therefore, that further scientific studies on the Fusarium wilt problem should also include analytic research upon these complex "unfavorable conditions," and also an investigation to determine why the cultivation of one crop may, in some way, be the ruin of the next one.

D. TUBER ROT AND RING DISCOLORATION OF THE POTATO

Of the older German works concerning injuries of potato tubers the best known are Martius (1842), Harting (1846) and Schacht (1856). These authors found Fusaria mixed with several types of tuber rot, but did not consider them as the proved cause of the disease. Martius thought there was a possibility that the Fusarium reduced the power of resistance of the tubers and predisposed them to decay.

The first artificial infections which proved conclusively that the fungus Fusarium caused a special tuber-rot of the potato were made by Pizzigoni (1896) and Wehmer (1897); the last by Pethybridge and Bowers (1908) and Longman (1909). Since the parasite was usually called *F. solani* it is possible that they were working with the same species. However, since negative results obtained by other scientists were interpreted in various ways, it seems doubtful whether this was the case (Frank, 1896, 1898). *F. solani* is considered as an obligate saprophyte by De Bary (1861), Reinke and Berthold (1879) and Roze (1897). Doubts as to its parasitic nature were expressed also in hand books and systematic works. Sorauer (1886) was more positive in his decision. Massee (1903, 1904) was sometimes uncertain, sometimes positive, while Lindau (1905, 1907) remains sceptical. This confusion was finally explained by the fact that *F. solani* was considered as a conidial form of an ascomycete. By Reinke and Berthold (1879) it is classified under *Hypomyces solani;* by Massee (1904) and Evans (1908) as *Nectria solani* (Pers.). The hyphomycete genera Cephalosporium and Monosporium are said to be other secondary fruiting formations in the development of the fungus (Massee, 1904).

F. solani (Mart.) is also mentioned in connection with diseases of other host plants, such as fruit rot of the tomato (Galloway, 1888; Elizabeth H. Smith,[5] 1907) and damping off of coniferous seedlings (Gifford, 1911). Elizabeth Dale[6] (1912) finally isolated the fungus from sandy soil along with other soil organisms, such as Ozonium, Mucor and *Verticillium alboatrum*, the latter of which she recently supposed to be the cause of "blindness" in potato tubers, a disease so-called on account of its completely destroying the eyes of the tubers. In the opinion of the author, this organism, however, is *Periola tomentosa* (Fr.), Reinke & Berthold (1879), a fungus closely related to Gloeosporium and only resembling Verticillium macroscopically.

[5] Conidia drawn in Elizabeth Smith's paper have the exact shape of young spores of *F. falcatum* App. & Wr. (1910).

[6] Ann. Mycol. **10**: 452–477. 1912.

The description of forms and colors of the fungus agrees with *F. rubiginosum* App. & Wr. (1910).

Ann. Bot. (London) **26**: 129–131. 1912.

Smith and Swingle (1903) report injuries to the growing potato plant caused by a Fusarium located in the vascular-ring of the mother tuber. They call it *F. oxysporum*, but do not separate it from *F. solani*, because, in the absence of morphological studies, they found it impossible to distinguish these species. By means of artificial inoculations, Manns (1911) confirmed the results of the last-mentioned authors, but undervalued the morphology of the parasite, an important factor for comparative studies, as will be seen. Thus, it is not surprising that Lounsbury (1909) should try to simplify the question by supposing that *F. solani* and *F. oxysporum* are identical and represent the conidial stage of *Nectria solani*, the cause of stem-end rot, white rot and winter rot.

Summarizing the results from the morphological standpoint, the literature presents an almost hopeless confusion. The morphology which should have been the basis of pathology has been neglected because of our insufficient knowledge of the taxonomy of the genus Fusarium (Link). From the pathological standpoint, however, we find that Pizzigoni, Wehmer, Pethybridge and Bowers, Longman, and more recently Jamieson and Wollenweber (1912) have successfully produced tuber rot artificially. The last authors have conclusively proved by artificial inoculations with the pure-grown fungus that Fusarium can entirely destroy the tuber. Among others, Smith and Swingle and Manns, the latter by means of inoculations, proved *F. oxysporum* to be the cause of a stem-end ring discoloration of tubers, and also the cause of wilt, or blight, of the growing plant. However, they do not separate fusarioses causing tuber rot (plate V, *c* and *e*) from those causing both the wilt disease of the plant and ring discoloration of the tuber (fig. *d*), so the reader might conclude that both wilt disease and tuber rot are caused by the same fungus.

According to the author's own investigations, the following different species are erroneously included in the earlier literature under the name *Fusarium solani* (Mart.): *F. Martii* App. & Wr. (Martius, 1842), fig. *b*; *F. coeruleum* (Lib.) (Hallier, 1898), fig. *c*; *F. rubiginosum* App. & Wr. (Schacht, 1856; Elizabeth Dale, 1910), fig. *g*; *F. falcatum* App. & Wr. (Elizabeth Smith, 1907), fig. *l*; *F. oxysporum* (Schlecht.) (Smith & Swingle, 1903; Manns, 1911), fig. *f*; *F. ventricosum* App. & Wr. (W. G. Smith, 1884), Fig. *q*; *Hypomyces solani* Reinke & Berth. (1879); *Nectria solani* (Pers.) (Massee, 1904; Evans, 1908); and *Ramularia didymum* (Hart.) n. n. (syn. *Fusisporium didymum* Harting, 1846), fig. *r*.

After having isolated some 500 strains of Fusaria, including 100 from potatoes (the potato is the host of 30 different forms, two-thirds of which are species, the rest varieties), the author has confidence that he can easily identify the above enumerated fungi.

This list of pseudonyms of *F. solani* (Mart.), even if not complete, shows, in connection with the illustrations of conidia (text figure) some striking contrasts. The first four species are described by Appel and Wollenweber (1910). The Hypomyces and Nectria, never found by the author himself, are harmless saprophytes according to De Bary, and Reinke and Berthold. The Nectria has, like *N. cinnabarina* (Tode) Fr., a Tubercularia-like stage but no sickle-shaped conidia, while the Hypomyces has a Fusarium stage which is more closely related to that of *Hypomyces ipomoeae* (Hals.) Wr. (fig. *d*) than to any other Fusarium. This fact was proved by comparative studies of various species of Hypomyces and Nectria, all being found cultivable in pure culture, in connection with routine determinative work on ascomycete material.

While we easily may leave out here both Nectria and Hypomyces, on account of their supposedly saprophytic nature, *F. oxysporum* (Schlecht.) is of special interest, being a serious enemy of American potatoes. The pathogenicity of this fungus, established by Smith and Swingle, has been confirmed by the author for the strain (fig. *f*) on which the diagnosis given above is based. The geographic distribution of the disease is based on hundreds of pure cultures of the fungus which were made and determined mainly from material collected by W. A. Orton. Comparisons of great value were made possible through the kindness of A. D. Selby of Ohio, who repeatedly sent tubers infected both with *F. oxysporum* and tuber rot Fusaria.

The diagnosis of *F. oxysporum* includes some additions to the description given by Smith and Swingle. These are the results derived from a special pure culture method. It also brings out the striking fact that the fungus, a typical xylem inhabitant (plate V, fig. *d*), does not entirely destroy the tuber without the help of tuber rot Fusaria or bacteria.

The fact that *F. oxysporum* causes the wilt of growing potato plants and only uses the xylem of the stem-end of tubers for over-wintering, without producing a rot of the parenchyma, leads to interesting comparisons with the following four species which are able to destroy the tuber entirely from artificial wounds, viz., *F. coeruleum* (Lib.), *F. trichothecioides* Wr., *F. discolor* var. *sulphureum* Schlecht. sub sp., *F. ventricosum* App. & Wr.

Methods. Before proceeding with the details of the inoculation experiments by which the parasitism of the above species was proved attention is directed to the methods employed. After the mechanical cleaning a careful selection of the tubers was made. Some tubers with slight specks on the skin and with Rhizoctonia pocks were used, however, when the supply was limited, providing the injuries proved insignificant when the potatoes were scraped. They were then washed and spread out once

more, in order to close up the wounds caused by cleaning.[7] After dipping them again in water the potatoes were arranged on perforated, galvanized iron plates with the stem-end turned up, and stored in a closed chamber in such a way that the formaldehyde could easily penetrate between the plates and reach the entire surface of the tubers.

Liquid formaldehyde is preferably used, in order to get a damp atmosphere and to keep the tubers wet during the treatment in the closed chamber. A higher percentage of tubers, fungus free but healthy, results from a long treatment (one to two days) in a formaldehyde weak atmosphere than from a short (one-half to one hour) treatment in a solution of $\frac{1}{3}$ to 1 per cent formalin. Seventy-five cubic centimeters of 40 per cent liquid formaldehyde suffice for each cubic meter of a large tight chamber. Formaldehyde, slowly heated, generates the gas at such a rate that a gradual but constant diffusion into the cork cover takes place. In this way the gas reaches the microscopically small air vacuoles in the deepest cork layers. This is important because the vacuoles are refuges for microörganisms. The only very resistant fungus which was not always killed in this treatment was *Periola tomentosa* (Fr.) Reinke & Berthold, a fungus often mistaken for Vermicularia and Verticillium in earlier literature.

The artificial injury of the tubers was performed by cutting off a stem-end segment of such a thickness as to guarantee tubers free from any spontaneous infection from the xylem ring. The danger of getting such a spontaneous infection from *F. oxysporum* is larger in America than in Germany, where *F. oxysporum* has not been found by the author during four years investigation, although it may, however, be found in southern Europe. At least, some Austrian authors, attribute a disease, a typical wilt disease according to descriptions, to fungi which cause symptoms similar to the *F. oxysporum* wilt. But they confuse it with a very distinct disease called Blattrollkrankheit.

Inoculation of the tubers was performed by sprinkling them with conidia suspended in distilled water. Large Altmann thermostats, which were built up in two series of ten chambers for temperatures from 0 to + 20°C. and from + 22 to + 60°C., served as inoculation chambers. The advantages of these lie in a uniform minimum of light, moderate air exchange

[7] If this is neglected, formaldehyde produces deep brown spots of dead parenchyma, as long as the gas acts. Only in open air, by normal transpiration, is the tuber enabled to isolate the poison center by corking it up and leaving depressions on the surface corresponding to hemispherical zones of killed parenchyma. When observing a cross section of the tuber, such spots appear sometimes as the first stages of a Fusarium rot, or of certain forms of brown-spot, which often occur in nature. The effect of formaldehyde is, therefore, a test as to whether the seed potato is suited for inoculation experiments, or whether the skin is wounded or breakable, although it appears microscopically sound.

through an opening closed loosely with cotton, the possibility of observation through the inner glass door, which must remain shut for a month in order to prevent secondary infections, the control of humidity by Lambrecht hygrometers, which are placed upright in the thermostats so that the scale can easily be read through the door. A slight disadvantage in the use of the chambers lies in the uneven temperature in one and the same compartment, which in spite of the double door, is in connection with the front, which is not quite hermetically closed and is subject to changes in humidity and temperature. Between 25 and 30°C. deviations up to 5°C. have been found within an individual chamber, with the minimum in the front near the bottom and the maximum near the ceiling at the back. The thermometers, read from the outside end, therefore, give the average temperature only when reaching the center of the chambers. The deviations, however, are of importance only for the cardinal points of the tuber infections.

Another valuable method for producing tuber rot artificially is that successfully tried by Jones, Giddings and Lutman (1912) in their work on Phytophthora. This consists in growing the fungus in the dark on living formalin-sterilized tubers placed in glass tubes. It is, of course, necessary that the tubes contain sufficient sterile water to maintain a damp atmosphere.

The experiments which the author began in Germany have been continued in Washington. The positive results repeatedly obtained show that the method is fit also for fruit and grain inoculations. Plate V, figures *a–c*, gives the arrangement of potatoes in the thermostats. All of the tubers except *b* were sprinkled after treatment with conidia of *Fusarium coeruleum* (Lib.). The check tuber cut and sprayed with sterile water (plate V, fig. *b*) and the unwounded, but infected, tuber (plate V, fig. *a*) remained healthy during the experiment, as indicated by the germination of the latter. Also the three tubers indicated at *c* bore healthy sprouts on the lower part after forty-three days, but these had no sooner died than the fusariose reached them. The xylem ring was not discolored more than the parenchyma, and the uniform brown discoloration of the fungus-infected part did not precede the advance of the fungus (plate V, fig. *e*).

By this method, *F. coeruleum* (Lib.) has been proved to be a wound parasite on the following potato varieties: Magnum Bonum, Up-to-date, Early Rose and Burbank. Every tuber was completely killed by the fungus within the limits of + 7 to + 30°C. within about two and one-half months, in the presence of at least 75 per cent atmospheric humidity. The higher the temperature the more rapid was the progress of the rot. From 7°C. down to the death point from frost, and from 30°C. upward to

38°C., there was a minimum of damage, the amount being a function of atmospheric humidity. Higher temperatures were of no importance, because the tubers could not withstand heat for a long period. It is interesting that the most progressive attacks of Fusarium take place within the temperature zones favorable for tuber germination. The same thermal relations which favor the Fusarium rot have been determined for the rot caused by Phytophthora (Jones, Giddings and Lutman, 1912).

The other above-mentioned Fusaria caused rot under the same conditions. However, *F. rubiginosum* App. & Wr., a fungus more frequently isolated from cereals than from potatoes, formed a contrast in only causing rot in the higher ranges of the thermal zone of the other fungi, and this very irregularly. This was not surprising because the fungus, a serious enemy of cereal seedlings, has a higher optimum temperature as indicated by the heavy losses it causes in parts of the United States of America having an average temperature five degrees higher than middle Europe. *F. rubiginosum* causes damping-off of oats and wheat, and to some extent barley, according to unpublished experiments performed by E. C. Johnson, and communicated by him to the writer. *Fusarium rubiginosum* is as wide-spread as *Gibberella Saubinetii* (Mont.) Sacc., which is another wound parasite of cereals, causing seedling blights and scab of grains (Selby and Manns. (1909). In contrast to *F. rubiginosum* it may be said that *F. subulatum* had a lower optimum than the other wound parasites; the slight damage it caused on potatoes might be understood by the fact that it is more common on cereals in middle Europe and probably there a cause of scab and seedlings blight like the others already mentioned.

Neither *F. solani* (Mart.), the most common cosmopolitan potato fungus, nor the majority of other species, could attack the wounded tuber at all. No one of all these saprophytes and wound parasites has the slightest relationship to *F. oxysporum* and *Verticillium albo-atrum*, the two causes of potato wilt. The fact (plate V, fig. *d*) that the latter cannot produce a tuber rot gives a biological contrast (plate V, fig. *e*) to the wound parasites of the tuber, and the fact that they cause the wilt disease of the growing plant, presents a contrast to the saprophytes.

SUMMARY

1. Variation in the formation of a stroma on different substrata prove this character to be unreliable for the classification of ascomycetes.

2. A culture method by which a normal growth of the fungi is secured on selected media has greatly aided in the differentiation of species.

3. With some exceptions, such as in Hypomyces, it will probably prove a general rule that the presence of chlamydospores in the true sense indicates the absence of an ascus stage.

4. The genus Fusarium is divisible into sections not only by physiological characters (pathogenicity) but also by morphological characters (conidia, chlamydospores).

5. One of the established sections, Elegans, comprises the vascular parasitic Fusaria, which are serious enemies of plants, causing especially wilt diseases.

6. Other sections contain more or less uniformly parasitic species; these cause fruit rot (Gibbosum and Roseum), tuber rot (Martiella, Discolor, Gibbosum, Ventricosum), scab and blight (Discolor).

7. Neocosmospora, an obligate saprophyte, is not genetically connected with the section Elegans of the genus Fusarium.

8. *Fusarium oxysporum* and *Verticillium albo-atrum* have no connection with leaf roll disease (the German Blattrollkrankheit), but cause stem wilt diseases with symptoms identical with the Kräuselkrankheit described by Reinke & Berthold.

9. Potato Fusarium tuber rot is caused by *F. coeruleum*, *F. discolor* var. *sulphureum*, *F. trichothecioides*, *F. ventricosum*, etc., while tuber ring discoloration is a secondary symptom of stem ring wilt disease (*F. oxysporum*, *Verticillium albo-atrum*, bacterial species).

10. Conditions favoring rapid infection are: A temperature of 14 to 26°C. in the presence of 75 per cent and more of atmospherical humidity, little ventilation, for both wilt and rot disease fungi; light sandy soil, hypocotyl inoculation for wilt; lack of light for rot disease fungi.

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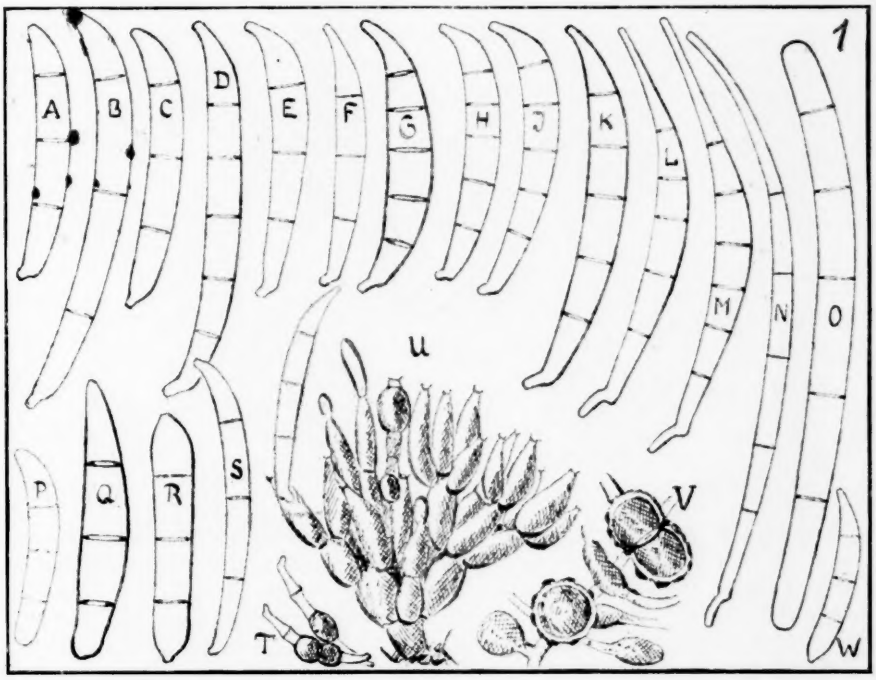

FIG. 1. The conidia in some sections of Fusarium show sharp contrasts between different sections, but a confusing similarity of form with the conidia of some ascomycetes. All grown in pure culture on steamed plant stems. Magnification × 1000, except *t* × 500.

a–c. Sectio Martiella: a, Fusarium solani (Mart. pr. p.); *b,* F. Martii App. & Wr.; *c,* F. coeruleum (Lib.).

d, Hypomyces ipomoeae (Hals.) Wr. (syn. Nectria ipomoeae Hals.).

e. f, (t, u, v) s. Sectio Elegans: e, F. redolens n. sp.; *f,* F. oxysporum (Schlecht.) *t* and *v,* chlamydospores. *t,* intercalated within conidia; *u,* conidiophore of the sporodochia-stage; *s,* F. orthoceras App. & Wr.

g–j, Sectio Discolor: g, F. rubiginosum App. & Wr.; *h,* F. discolor var. sulphureum Schlecht. (s. sp.); *j,* F. trichothecioides Wr.

k, Gibberella Saubinetii (Mont.) Sacc.

l, m, Sectio Gibbosum: l, F. falcatum App. & Wr. *m,* F. gibbosum App. & Wr.

n, Sectio Roseum: F. subulatum App. & Wr.

o, Nectria galligena Bres.

p, Neocosmospora vasinfecta Smith.

q, Sectio Ventricosum: F. ventricosum App. & Wr.

r, Ramularia didymum (Hart.) n.n. (Syn. Fusisporium didymum Hart.). This conidium is an exception to the rule of 1-septate conidia, but normal in shape.

w, Calonectria graminicola (Berk. and Brm.) Wr. n. n. (Syn. Nectria graminicola Berk. & Brm.).

EXPLANATION OF PLATE V

FIGS. *a–c*. Dry rot of potato tubers artificially produced by sprinkling the cut surface of the potatoes with conidia from pure cultures of wound parasitic Fusaria. Time of exposure, forty-three days; thermostat + 16 to + 18°C.; atmospheric humidity 75 to 85 per cent. *a*, tuber not wounded but infected with F. coeruleum; *b*, tuber cut at the stem end, sprayed with water without spores; *c*, three tubers, cut at the stem end and sprayed with conidia of F. coeruleum.

FIGS. *d–e*. Ring disease and rot of the potato tuber seen from longitudinal sections. *d*, stem end ring disease, a secondary symptom of the Fusarium wilt disease of the potato plant, caused by F. oxysporum. No cause of rot, except in association with tuber rot Fusaria or Bacteria; *e*, typical tuber rot showing the uniform progress of rot through the whole parenchyma.

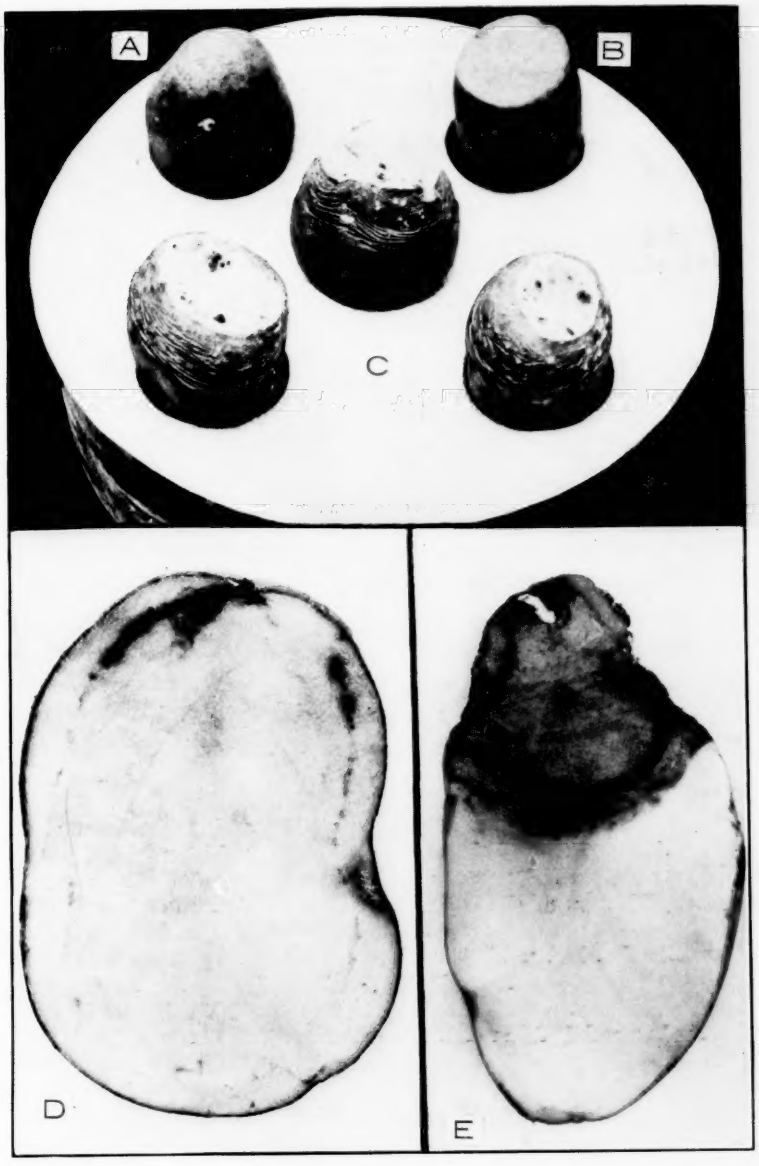

PLATE V. STUDIES ON THE FUSARIUM PROBLEM

SEPTORIA PISI IN RELATION TO PEA BLIGHT

I. E. Melhus

With Plate VI

The investigation of *Septoria pisi* (West.) in its relation to pea bligh was started in June, 1911. At this time the Wisconsin Experiment Station was receiving many inquiries as to the cause of certain damages to the pea crop in all sections of the state. Most of the growers sustained a loss of 25 per cent and in some cases whole fields were a total failure. The damage to the crop manifested itself as follows: (1) in the destruction of some or all of the diseased plants early in June, following a spell of wet weather; (2) in injury to the stems of plants less severely attacked so that the pods did not fill or so that the plants wilted down and dried up during the hot weather in the early part of July, before the pods had reached sufficient maturity to be used for canning purposes.

The plants killed early in June did not seem seriously infected, judging from the appearance of the leaves, but the lower third of the stems was blackened and shrunken. In some cases, however, pycnidia of Septoria were present on the lower diseased leaves and petioles, but they were not abundant, apparently due to the death of the plants before the fungus had time to fruit. In the majority of cases the stems were more severely attacked at the nodes. The nature of such an infection is clearly shown in plate I, figures 4 and 6. A plant taken from a field so badly blighted that it was not harvested is shown in plate VI, figures 1 and 2. The lower leaves and the basal portion of the stems were in every case thickly sprinkled with mature pycnidia of Septoria. The general effect on the plant is evident from the number and size of the pods.

It is well known from the investigations of Hiltner,[1] Krüger,[2] Jarius[3] and Van Hook[4] that *Ascochyta pisi* may cause severe damage to the pea, much resembling that described above. However, in these cases

[1] Hiltner, L. Über die durch Ascochyta pisi hervorgerufene Wurzelkrankheit der Erbsen. Centbl. Bakt., Abt. 2, **1**: 881. 1895.

[2] Krüger, F. Ungewöhnliches Auftreten von Ascochyta pisi Lib. an Erbsenpflanzen. Centbl. Bakt., Abt. 2, **1**: 620. 1895.

[3] Jarius, Max. Untersuchungen über Ascochyta pisi bei parasitischer und saprophytischer Ernährung. Bibliotheca Botanica **34**: 1–21. 1896.

[4] Van Hook, J. M. Blighting of field and garden peas. Ohio Agr. Exp. Sta. Bul. 173: 231. 1906.

Ascochyta was not present in any quantity, but another imperfect fungus, *Septoria pisi*, which has not, heretofore, been reported as very destructive, was associated in great abundance. This led me to take up the study of Septoria in order to learn whether it might cause such symptoms as were noted in the field, as well as to learn its pathogenicity and distinguishing characters, so that it might not be confused with *Ascochyta pisi*.

Artificial infection experiments. Pea plants of all ages and of several varieties were subjected to infection, at different times, from July, 1911, to October, 1912. A large number were infected with uniform results so it is not necessary to relate the details of the numerous experiments at this time. The plants used were grown in 6-inch pots or in small flats. These were infected by spraying with a suspension of spores, after which the plants were incubated at 22 to 24°C. in a saturated atmosphere for twenty-four hours. Especial effort was made to infect pea seedlings from 2 to 6 inches high, since it is in this stage that the greatest damage is done in the field. Infection developed usually in nine to twelve days, although sometimes it required a longer period. The variation in time was doubtless due to environmental conditions, such as moisture and temperature. The first indication of infection is the development of light-green irregular areas, which may occur anywhere on the leaves, but more often at the margin of the leaflets, due to the collection of droplets of water at this point. Gradually, the color of the infected areas changes from light- to yellowish-green, finally becoming light-brown, due to the death of the infected tissue (pl. VI, fig. 5). There is no tendency to form the dark spots so characteristic of Septoria on various other hosts. Instead of this the fungus spreads outward from the initial point of infection until it has included the entire leaflet. It fruits very soon after the infection is well established in the host tissues, as is evident from the photograph reproduced in plate VI, figure 5. The first indication of pycnidia appears about four days after the infection is evident, or from nine to twelve days after the plants are exposed. At this time the surface of the infected area is rough and irregular and on the fifth day the pycnidia have come through the epidermis and are plainly visible. In this stage of their development they are light-yellow in color and filled with spores. As they become older they gradually change from light-yellow to brown and finally become black.

The fungus does not stop after destroying the leaflet; it has only begun its destructive work. It enters the petiole and travels down into the the stem. It is here that Septoria does its damage, especially if the plant is young when attacked. The infection kills the stem tissues, thus interfering materially with the normal functions of the plant. If the infection is sufficiently vigorous it kills the host in a few days, otherwise the plant

may remain alive for some time, dying gradually. A large proportion of the infected plants go on to maturity and materially reduce the yield, which depends, of course, upon the extent and age of the plants when infected. The pycnidia of Septoria do not appear as quickly on the stems as on the leaves and only develop when the host plant begins to mature. At this time it is not unusual to find the fungus fruiting on the lower half of the stems, as is shown in plate VI, figure 1. My infection experiments show plainly that *Septoria pisi* attacks and kills both the leaves and stems of the pea when artificially infected and that the greatest injury results from stem infection. Symptoms like those found in the blighted fields have been produced, which suggests that the damage done in the open may well have been due, in part at least, to the Septoria.

Outdoor infection experiments. In order to study the effect of Septoria under more normal conditions than was possible in the greenhouse, experiments were conducted in the pathological garden. Plants 6 inches tall were exposed to infection in the manner already described, but without positive results. This was not at all surprising because the weather from July 14, 1911, the time the plants were exposed to infection, until July 27 was very dry and hot, a condition under which Septoria does not flourish in the field.

It was thought advisable to approximate field conditions more closely and allow infection to take place by natural agencies. For this purpose three plots, each 6 feet square, were selected and prepared for planting. Two of these plots adjoined and the third was about 5 rods distant. In one of the two adjoining plots, which I shall call A, pea vines were partially buried in a manner comparable to plowing. These pea vines were taken from a market garden in the vicinity of Madison where Septoria was very abundant and destructive. Pycnidia were very numerous on these vines and examination showed that many were still filled with viable spores. Alaska peas were planted in the three plots on August 8. The rain of the following night made conditions immediately favorable for the peas to germinate, and in six days they were up. On August 21 infections began to appear on the lower leaves, which became yellowish-green in irregular areas and finally light-brown, due to the death of the tissues. This infection was identical with the one resulting from the exposure of pea plants to Septoria in the greenhouse. The infection spread throughout the leaf tissue in the way already described, and when the leaflets had been destroyed the fungus attacked the petioles and traveled down into the stem.

Careful count of the plants in plot A on August 24 showed that 90 per cent were infected. Early and abundant infection was undoubtedly favored by the damp moist weather that followed several days after the peas

came up. It was especially interesting to note that on the first three rows of plot B, adjoining plot A, the amount of infection was about the same, but in the remaining rows of plot B there was less, possibly only 75 per cent. This suggested that the source of infection was the dead pea vines in plot A. It is also probable that the wind was the chief agency in distributing the spores. Careful examination of control plot C, 5 rods to the south, beyond several rows of corn and tobacco, showed no evidence of Septoria previous to August 24. This fact further shows that the source of the spores was the infected pea vines.

Observations made on August 29 showed little or no leaf infection on any of the plots. The infected leaflets had been killed and dried up and the only evidence of infection at this time was on the stems, especially near the surface of the soil. These were discolored and shrunken at the nodes where the diseased leaves had been attached. Some of the plants were already dead, while the rest were less vigorous than the healthy ones in the control plot. New infection of the younger leaves had been checked by the hot dry weather that intervened between August 18 and 29. Septoria pycnidia containing mature spores were found September 5 on infected petioles. This further established the fact that Septoria was the fungus causing the damage and that the infection might take place through natural agencies.

No further results worthy of mention developed until September 18, when an outbreak of Ascochyta occurred. The vines at this time were nearly a foot tall and lay semi-prostrate on the ground. The source of this infection was doubtless a small plot of peas less than two rods distant that were infected with Ascochyta earlier in the season, and which had only recently begun to fruit, so that they were well covered with mature pycnidia. Ascochyta developed on all three of the plots, although not so extensively on the check plot. This was doubtless due to its greater distance from the vines producing the Ascochyta spores. As I shall explain later, the early symptoms of Ascochyta are distinctly different from those of Septoria so this outbreak led to no confusion. It was not until September 23 that I was able to find any new Septoria infections and these were not numerous. This decrease in the amount of Septoria I attributed to the lack of spores, as the supply contained in the pea straw had functioned earlier and the fungus had not had sufficient time to fruit extensively.

In order to gain additional evidence, the experiment was repeated. Septoria-infected pea vines that had been collected on the same date as those used earlier and kept in the laboratory were partially buried, as in the previous experiment. Peas were planted August 22, two weeks later than in the preceding experiment. Septoria came on in much the

same way as already noted, so it is needless to describe the results further. It is clearly shown that infection can be obtained on pea plants in the open and that symptoms like those found under field conditions develop. These facts, coupled with many observations, make it quite conclusive that Septoria was an important factor in the damage done by pea blight in Wisconsin during the season of 1911.

Symptoms of Septoria and Ascochyta. The symptoms of Septoria on the pea have never been clearly defined and it is reported by Halsted,[5] and Stevens and Hall[6] that Septoria and Ascochyta can not be distinguished from one another on the host without the aid of the microscope. This is very true in certain late stages of the two diseases, but the early symptoms are quite unlike and are readily distinguishable. For instance, it is known from the results of Krüger,[7] Van Hook[8] and my own studies that Ascochyta produces spots with ashen-white centers surrounded by dark borders. Septoria, on the other hand, produces no distinct spot and the first indication of infection is the appearance of yellowish-green irregular areas, which later become light-brown, due to the death of the tissues. Primary infection with Septoria seldom, if ever, occurs directly on the stem, while this is a very common occurrence with Ascochyta. Septoria reaches the stem at the nodes by attacking the petiole and stipules, as is shown in plate VI, figures 4 and 6. If the infection occurs early the plant may become girdled (pl. VI, fig. 6). Ascochyta causes pronounced lesions anywhere on the stem, which may, and usually do, coalesce and girdle the plant if the infection is severe. Late stages of stem infection, both before and after the fungi have fruited, are not readily distinguishable.

Mycosphaerella pinodes (B. & Blox.) Johans. On October 24 some of the dead vines from plot A were brought into the laboratory and examined for Septoria and Ascochyta. Both fungi were present, as well as a small ascomycete. On the same day another collection was made from the plot that was so generally infected with Ascochyta earlier, and perithecia were found to be very abundant. Most of them were immature, but a few were found that contained mature spores. The asci were short and eight-spored. The two-celled spores were constricted in the middle and somewhat pointed at each end, with prominent oil droplets in each cell. The average of sixteen measurements showed them to be 16 x 6.5 microns.

This is doubtless the species listed by Cook[9] as *Sphaeria pinodes* (B.

[5] Halsted, B. D. Some fungous diseases of the pea. New Jersey Agr. Exp. Sta. Rept. **1893**, p. 357.

[6] Stevens and Hall. Diseases of economic plants, pp. 255–259. 1910.

[7] Loc. cit.

[8] Loc. cit.

[9] Cook, M. C. Handbook of British fungi **2**: 909. 1883.

& Blox.), although there is a little difference in the spore measurements. Later, the subgenus Sphaerella of Fries was raised to generic rank by Cesti and de Notaris, and Saccardo[10] subsequently lists two species of Sphaerella, viz., *S. morieri* (Crie) Sacc., occurring on the dead leaves, and *S. pinodes* (B. & Blox.) Niessl., occurring on the stems. The descriptions of these two species are otherwise quite alike and it is not at all improbable that they represent the same fungus. Grove[11] has recently shown that Sphaerella was established in 1824 by Sommerfelt for a group of algae and is not correctly used as a fungus genus, and that the name Mycosphaerella, which was established by Johannson in 1884, should be used as the name of the fungal genus. It is suggested by Saccardo that *Mycosphaerella pinodes* may be the perfect stage of *Septoria pisi*. Potebnia,[12] who has collected and studied *Septoria pisi* in Russia, is, likewise, of the same opinion. However, the following experiment tends to show that such is not the case. Single spores of this ascomycete grown in pure culture on potato agar produce Ascochyta pycnidia. The spores were isolated by the dilution method and individual ones were located and transferred to other plates. Six spores were thus obtained, all of which produced Ascochyta in pure culture, except in one case where there was bacterial contamination. Spores taken from these pure cultures of Ascochyta and used to infect young pea plants produced Ascochyta lesions in quantity, but no Mycosphaerella developed on the plants, even after they were dead. Thus, the evidence was not conclusive that Mycosphaerella is the perfect stage of Ascochyta. It should be noted, however, that the failure to obtain Mycosphaerella on Ascochyta-infected plants in the greenhouse should not be construed as demonstrating positively that no relation exists between the two fungi. The abnormal environmental conditions in the greenhouse may well prevent the formation of the perfect stage.

It was next thought advisable to isolate immature perithecia and grow them in pure culture. This was done, and Ascochyta again developed. This test was also very suggestive as to the relation of the two fungi concerned. In view of the fact that Mycosphaerella appears to be related to Ascochyta rather than Septoria, further study of it was discontinued by the writer and taken up by R. E. Vaughan, who is making an extensive study in this laboratory of pea blight caused by Ascochyta.

It is not known how Septoria passes the winter, although my results and observations tend to show that it must have a perfect stage which functions in this capacity. That the pycnospores can not perpetuate the

[10] Saccardo, P. A. Sylloge Fungorum **13**: 877.

[11] Grove, W. B. Sphaerella v. Mycosphaerella. Journ. Bot. **50**: 89–92. 1912.

[12] Potebnia, A. Beitrage zur Micromyceten Flora Mittel-Russland. Ann. Myc. **8**: 70. 1910.

disease from year to year is quite conclusive. Infected pea vines, which contained large quantities of viable spores at the time of collection in June and July, were stored in the laboratory and from time to time the germinating capacity of the spores was tested. Their viability decreased until in February none germinated. Again, it might be argued that Septoria infects the seed and perpetuates itself from one year to another in much the same way as Ascochyta is known to do, but this is not in accordance with my observations and experiments. The pods become abundantly infected with Septoria (plate VI, fig. 2), but the mycelium does not penetrate through the pod into the seeds. Peas have been soaked for twenty-four hours and then exposed to Septoria infection by spraying with spores and subjecting them to favorable conditions for the growth of the fungus. In no case has infection of the seed resulted. Pea vines known to have been infected in the late summer and fall with Septoria have also been examined for Septoria spores in the spring, but in every case the pycnidia have been found empty. Viable spores have been placed in water on slides and subjected to temperatures varying from 0 to −10°C. for twenty-four hours and then returned to the laboratory and subjected to favorable conditions. In every case the spores failed to germinate. Similar spores dried on slides and subjected to low temperatures have also refused to germinate when favorable conditions were restored.

It is not improbable that *Septoria pisi* has a perfect stage which is instrumental in starting infection in the spring. It was observed that the fields became very generally attacked early in June when the peas were from 2 to 6 inches high, depending upon the time of planting and weather conditions. If the fungus propagated itself through the seed one ought to find certain areas in the field more generally infected than others, during the early development of the disease, but such is not the case. On the other hand, I have examined whole fields and found the infection uniform, suggesting that spores were either produced locally, or at some distance and distributed by the wind. Several ascomycetes occur on the dead straw but their relation to Septoria is not known at the present writing.

Cultural studies. Septoria has been grown in pure culture by Potebnia,[13] who found that it produced conidia and dense balls, but no further details regarding its cultural characteristics are given. I have obtained the fungus from young infections in the leaves and from single spores isolated by the plate dilution method, but have not been successful so far in getting it to produce normal pycnidia, although it has been grown on a wide range of media. It grows slowly, and especially so, immediately after it

[13] Loc. cit.

has been obtained from the host tissues, but after it has been transferred a few times it develops more rapidly. In this respect it behaves in the same way as *Septoria pyricola* studied by Duggar. The submerged mycelium is dark colored and tends to form a thick crust which is covered by a thin white layer of mycelium. In the aërial mycelium are found stromatic masses which also become dark colored and have the general appearance of pycnidia, but so far none of them have been found to contain spores. These empty pycnidia tend to form more abundantly when the culture begins to dry out. The two-celled conidia are formed on side branches of the hyphae and have the same dimensions as the pycnospores. It should be noted, however, that conidia formation has been secured only in cultures several months old. Whether there is some correlation between moisture content and conidial formation is not known, but my results are suggestive. It has already been pointed out by Stevens and Hall[14] that colonies of *Septoria lycopersici*, grown under crowded conditions, produce naked spores, while, otherwise, pycnidia are formed in great abundance.

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EXPLANATION OF PLATE VI

FIGS. 1, 2, 3, 4. Plants infected with Septoria pisi taken from badly diseased fields.

FIGS. 1, 2. A plant killed by Septoria; 1, the fungus fruiting on the lower portion of the stem; 2, the upper portion of plant shown in Figure 1—note the absence of Septoria and the small pod.

FIG. 3. Septoria infection and pycnidia on pod and petiole.

FIG. 4. A stem infected at the node with Septoria; primarily caused by leaf infection. Magnified four diameters.

FIGS. 5, 6. Symptoms of Septoria produced by exposing young pea plants to infection in the greenhouse; 5, early stages of leaf infection; 6, late stages of leaf and stem infection.

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[14] Stevens, F. L. and Hall, J. G. Variation of fungi due to environment. North Carolina Agr. Exp. Sta. Rept. **32**: 47. 1909.

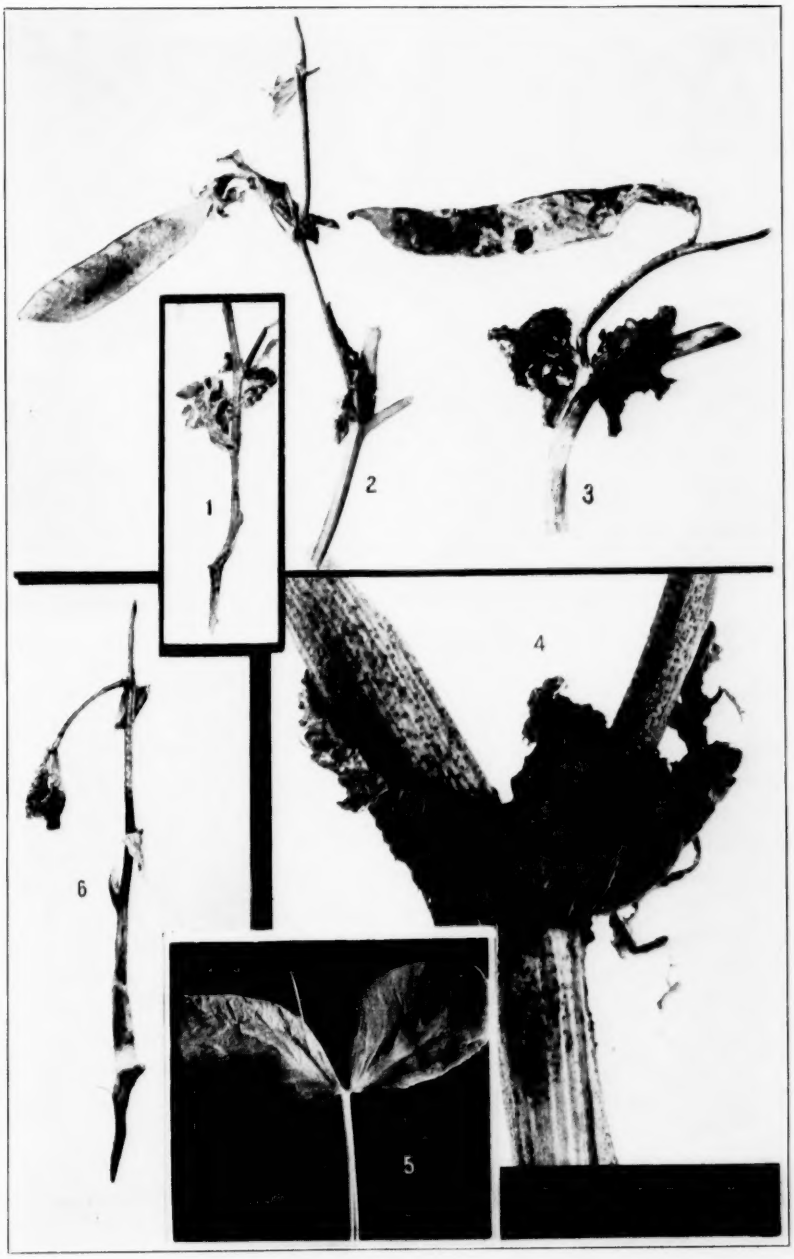

PLATE VI. PEA BLIGHT

SOME SUCCESSFUL INOCULATIONS WITH THE PEACH CROWN GALL ORGANISM AND CERTAIN OBSERVATIONS UPON RETARDED GALL FORMATION

CLAYTON O. SMITH

The wide range of hosts susceptible to infection with *Bacterium tumefaciens* has been well established through the work of Dr. Erwin F. Smith and his associates. Certain hosts that have never before been reported as susceptible have been successfully infected at our laboratory by artificial inoculations. The following is a list of plants upon which galls have thus been artificially produced:

Anacardiaceae: *Schinus molle*, pepper tree.

Ebenaceae: *Diospyros kaki*, Japanese persimmon.

Juglandaceae: *Juglans californica*, Southern California black walnut; *J. californica* var. *hindsii*, Northern California black walnut; *J. cinerea*, Butternut; *J. nigra*, Eastern black walnut; *J. regia*, English walnut; *J. sieboldiana*, Japanese walnut; *Hicoria pecan*, pecan.

Myrtaceae: *Eucalyptus tereticornis*, Forest red gum.

Rosaceae: *Cydonia* sp., Angiers quince; *Prunus amygdalus*, hard-shell and bitter almond; *P. armeniaca*, apricot; *P. avium*, mazzard cherry; *P. allegheniensis*, Allegheny plum; *P. davidiana*, ornamental almond; *P. domestica*, prunes, vars. Clyman, Yellow Egg, Sugar, Tragedy, French, Hungarian; *P. cerasifera*, vars. Myrobalan, Marianna; *P. mahaleb*, Mahaleb cherry; *P. orthosepala*, Texas plum; *P. persica*, peach, varieties Lovell, Muir, Salway; *P. platycarpa*, Peen-to or Saucer peach; *P. simonii*, Simon plum; *P. triflora*, plum, vars. Burbank, First, Kelsey, Satsuma, Wickson; *Pyrus betulifolia*, Chinese pear; *Py. communis*, French pear stock; *Py. pashia*, Chinese pear; *Laurocerasus lyonii* (*Prunus integrifolia*), Catalina cherry.

Rutaceae: *Citrus aurantium*, Valencia orange; *C. vulgaris*, sour orange; *C. limonum*, Eureka lemon; *C. limetta*, sweet lime.

Sterculiaceae: *Sterculia diversifolia*, Victoria bottle tree; *St. acerifolia*, flame tree.

Urticaceae: *Ficus carica*, fig.

Markedly retarded gall formation has been observed in two instances, that of Angiers quince and fig. In both cases the inoculations were made after rapid growth had ceased.

Angiers quince. A vigorously growing sprout of the current year's growth was puncture-inoculated on June 3, 1911. Up to January 23, 1912,

no galls had been found and no further observations were recorded until July 16, 1912, when small knot-like galls from one-sixteenth to one-eighth of an inch in height were found to be forming in the healed-up tissue of the old scars. Further inspection of these galls on October 15, 1912, showed that they had increased slightly in size and were becoming rougher or more warty. This tree has also what is believed to be the aërial quince knot, so common on quince trees in California, but these knots seem to differ from the artificial galls, both in manner of growth and shape. No natural knots have been found on the inoculated branch, nor do they appear until the wood becomes several years old.

It is interesting to note that similar retarded gall formations were observed by Dr. Erwin F. Smith and his associates in their inoculation of quince seedlings with cultures of the hairy root organism.

Fig (*Ficus carica*). A fig tree was inoculated on two branches August 24, 1910. Observations were taken at various times, as the tree was easily accessible, but no galls developed until early in 1912. At first there were only slight prominences that could scarcely be taken for galls. The particular branches that were inoculated had not been growing well and to stimulate new growth they were cut off just beyond the points of inoculation. On February 22, 1912, one branch showed a large gall and several smaller ones. The other inoculated branch showed, on June 15, 1912, one small gall. Growth in the part inoculated seems to be a necessary condition for the formation of galls, but the organism is able to live in the tissue for a considerable period of time (in this case one and one-half years) without apparent gall formation.

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ENDOTHIA RADICALIS (SCHW.)

C. L. Shear

In a previous note[1] we expressed the opinion that *Endothia radicalis* (Schw.) was based on the long ascospore form illustrated by Ellis and Everhart[2] as *E. gyrosa* (Schw.). This according to our present information appears to be an error, unless Schweinitz mixed two collections under this name. Only one collection, however, is mentioned in his published works. The specimen in Schweinitz's Herbarium at Philadelphia appears to show only pycnidia and we judge from Farlow's published statement that the one in his collection is in the same condition. There is in the Kew Herbarium an autograph specimen from Schweinitz, apparently on oak roots, which shows perithecia and ascospores. This is evidently the specimen which Schweinitz sent Hooker, and which was described and illustrated by Curry.[3] A slide from this specimen show ascospores 6.3 to 8.6 x 2.8 to 3.6μ, agreeing very closely in appearance with the two upper spores in Curry's figure. The two lower spores with two to three septa shown in his figure are unlike anything we have found on our slide from this specimen and they may belong to something else. The specimen of Schweinitz's species which should be regarded as the true type, however, is that sent by Schweinitz with his diagnosis to Fries and used by him in his description in Elenchus Fungorum. This specimen is preserved in Fries' Herbarium, is apparently on oak roots, and shows perithecia; but no measurements or description of the spores from this specimen have ever been published. The Kew specimen appears practically identical with *E. virginiana* And.

In the article referred to we also said that *Diaporthe parasitica* was probably introduced into this country from Europe. This statement was based chiefly upon the results of inoculation experiments made with material kindly given us by Dr. Pantanelli, of Rome, as of Italian origin, and which produced typical chestnut-blight cankers in the inoculation experiments referred to. We have since learned from Dr. Pantanelli that the material which he gave us was probably *D. parasitica* which he had in the laboratory at the time and which was given us by mistake for the Italian fungus. Other inoculations with the native Italian Endothia have not yet produced the disease.

[1] Phytopathology, **2**: 211. Oct. 1912.
[2] North American Pyrenomycetes, pl. 30. Figs. 6–8. 1892.
[3] Transactions Linnaean Society, **22**: 272. Pl. 47. Fig. 89. 1858.

PHYTOPATHOLOGICAL NOTES

Notes on Cronartium comptoniae. While making observations upon *Cronartium comptoniae* Arthur within a rather limited area counts were made of recently killed trees of *Pinus rigida* which bore signs of the attacks of this fungus. Within this area 147 living trees were found bearing the fruiting bodies of *Cronartium comptoniae* and 8 trees were found which had evidently died within two or three months and which evidently had been attacked by the fungus. That is, there was an evident annual mortality of approximately 5 per cent from the attacks of the fungus. Fires have had no effect within this area, as young pines were planted there several years ago and fire has been kept out since that time. The observations were made shortly after the uredo stage began to fruit and thus an excellent chance was given to determine the approximate distribution of the aecidiospores. It was found that the uredospores were distributed (in this case) in a definite direction from the diseased pines. That is, the distributing wind seems to have been fairly constant and the uredo stage, in each case, covered a fan-shaped area which had the infecting pine at the apex. The uredo stage at that time was practically all within ten yards of the point of infection. Undoubtedly, an extra hard wind or a small whirlwind might have taken the aecidiospores much further. The evidence shows that the great mass of aecidiospores is ordinarily blown but a short distance.

During the summer of 1912 the writer received young trees of *Pinus sylvestris* and *P. ponderosa* diseased with a Peridermium on the stems. Inoculations were made in the open upon *Comptonia asplenifolia* where *Cronartium comptoniae* has never been seen for over ten years. These inoculations were successful upon the inoculated leaves, which were enclosed in waxed paper bags, and in no case was the fungus found except on the inoculated plants. The uredo stage developed about two weeks after inoculation and several weeks later the teleuto stage also developed. So far as the writer knows this is the first time the transfer has been made from *Pinus ponderosa* to Comptonia.

PERLEY SPAULDING.

Winter injury to the white elm. During August, 1912, the attention of the writer was called to a general diseased condition of trees of *Ulmus americana* in Rockford, Illinois. An inspection of certain areas of the

city showed a very unthrifty condition of many of the trees, associated with a killing back of the top and, in many cases, a sloughing off of the outer bark nearly to the cambium. Limbs were frequently killed back for a distance of 10 to 15 feet, but bore no signs of parasitic fungi, although an occasional saprophyte was found.

The sloughing of the bark was the most interesting feature. This was limited, in some cases, to small areas at the base of the trunk, but in many other instances, the loosened layers, which could only be located by tapping the bole, could be stripped off the trunk, either partly or completely, to a height of 10 to 15 feet, or more. The cambium, except in an occasional very limited area, was living. The cleavage plane just outside the cambium usually appeared peculiarly sculptured, apparently due to growth phenomena after the injury had occurred. Since the loosened bark has persisted on the trunk for a season or more it bore considerable evidence of the activity of small bark borers. Larvae of the elm borer however, were scarce, and little indication of their presence was met with. Although a member of the Agaricaceae (*Marasmius* sp.) was found abundantly fruiting on the outer bark, at or near the base of many trees, an investigation did not reveal a causal relation of this organism to the trouble.

In addition to these distinct phenomena of limb and bark injury, other elm trees only showed decreased vigor, coupled with a yellowing of the foliage and early leaf fall.

With proper attention to culture methods, and a pruning out of dead limbs, the trees will probably gradually recover, in most instances, unless nature interferes again too unfavorably.

During pruning operations it is very essential that all freshly-cut surfaces be treated with an antiseptic coating in order to prevent the entrance of saprophytic decay organisms. Asphaltum has been highly recommended as a wound dressing for trees, but in lieu of this a thick lead paint will serve the purpose well.

C. J. HUMPHREY.

Personals. Mr. C. R. Orton, lately assistant botanist in the Indiana Experiment Station, has been appointed botanist of the Pennsylvania Experiment Station. Miss Adeline Ames has accepted a position as assistant forest pathologist in the Bureau of Plant Industry. The address of Dr. Ernest Shaw Reynolds, stated erroneously in the last number of PHYTOPATHOLOGY, is the North Dakota Agricultural College, Agricultural College, N. D. Dr. Ormond R. Butler, formerly instructor in horticulture in the University of Wisconsin has been appointed botanist in the New Hampshire Agricultural College and Experiment Station. H. E. Truax has been appointed assistant plant pathologist in the Arkansas University

and Experiment Station. R. A. Studhalter, recently assistant in botany in the Kansas Agricultural College, has been appointed agent in forest pathology in the Bureau of Plant Industry. His address is Zoological Building, University of Pennsylvania, Philadelphia, Pa. Orlo A. Pratt has been appointed assistant in plant pathology in the Idaho University and Experiment Station.

CLEVELAND MEETING OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

The fourth annual meeting of the Society was held in Cleveland, Ohio, December 31, 1912, to January 3, 1913, in conjunction with the American Association for the Advancement of Science.

Over fifty members were in attendance and a program of fifty papers was presented. The total membership at present is 240. Seven new members were elected at this meeting. Joint sessions were held with Section G of the American Association for the Advancement of Science and also with the Botanical Society of America.

The following officers were elected for 1913:

President—F. C. Stewart, New York Agricultural Experiment Station, Geneva, N. Y.

Vice-President—Haven Metcalf, U. S. Department of Agriculture, Washington, D. C.

Secretary-Treasurer—C. L. Shear, U. S. Department of Agriculture, Washington, D. C.

Councillor—W. J. Morse, Maine Agricultural Experiment Station, Orono, Maine.

The Society decided to hold its next annual meeting at Atlanta, Georgia, in conjunction with the American Association for the Advancement of Science.

The present mode of nomination of officers was adopted for the coming year. It was also decided that subscriptions from institutions or libraries cannot be substituted for membership fees of individuals. The Secretary was instructed to drop from the list of members those more than one year in arrears.

At the request of the Botanical Society of America a committee consisting of the newly elected President and Secretary was appointed to consider questions of closer affiliation with the two Societies. Upon motion the President designated the Secretary of the Society to act as its representative to meet with the Secretary of the American Society of Naturalists and other related Societies to consider closer affiliations, especially as to places of meeting and arrangement of programs.

F. C. Stewart was added to the Committee on Common Names of Plant Diseases and made Chairman of the same, the other members being F. L. Stevens, H. von Schrenk, E. M. Freeman, and G. P. Clinton.

The Secretary was instructed to publish the constitution of the Society and a list of the present members and also present and past officers of the Society.

The following resolutions were adopted by the Society:

Resolved, That the American Phytopathological Society, recognizing the fact that plant diseases do not recognize national limits or geographical boundaries and the evident limitations imposed upon investigations when restricted by national boundaries, respectfully recommend that administrators of research institutions, whether state or national, as well as individual investigators, recognize the importance of establishing closer international relations and take such steps as may be practicable from time to time to secure this end, including not only more frequent visits of American investigators to foreign countries for field observations as well as research, but also the securing, either by permanent or temporary engagement of the best of foreign experts in plant pathology.

Resolved, that the Society express its sincere thanks and appreciation to all concerned for the courtesies and accommodations extended to it at the Cleveland meeting.

A Committee on Resolutions regarding the death of Prof. M. B. Thomas, consisting of Donald Reddick, L. R. Jones, and W. A. Orton, was appointed. The following resolution was prepared by the committee:

MEMORIAL RESOLUTION OF JANUARY 3, 1913

Professor Mason Blanchard Thomas, Rose Professor of Biology in Wabash College, died March 6, 1912, in the forty-sixth year of his age.

At its first meeting after his death, this Society desires to inscribe upon its records an acknowledgment of the great loss this Society has sustained by the death of Professor Thomas, a scholar, a man of inspiring personality, an enthusiastic teacher of botany and an energetic supporter of our Society.

Professor Thomas was graduated from Cornell University in 1890 with the degree of Bachelor of Science and was the same year appointed Fellow in Botany in that institution. The following year he was appointed Professor of Biology at Wabash College, a position which he held until his death. Professor Thomas was deeply interested in his profession and took an active interest in the scientific meetings. He was a fellow of the American Association for the Advancement of Science, at one time a member of the council, a member of this Society, an active member and one time president of the Indiana Academy of Science, Vice-President of the Indiana State Forestry Association and a member of the honorary Societies of Sigma Xi and Phi Beta Kappa. He was honored by Wabash College June, 1907, with the degree of Doctor of Philosophy.

The published works of Professor Thomas are not numerous but his keenness for investigation is well known to more than two score young men who have entered the profession under his guidance and tutelage, while his optimism and enthusiasm for things worth while are known to many scores of students.

This expression of esteem for a colleague cut off in the midst of his achievements and his plans is inscribed with sorrow upon the minutes of this Society.

DONALD REDDICK,
L. R. JONES,
W. A. ORTON,
Committee.

Resolved, That the matter of the form of papers to be presented at the next meeting, whether in full or by abstract and discussion, be referred to the Council with power to act and the privilege of taking a referendum by mail, if deemed advisable.

In accordance with this resolution, the Council decided that all members offering papers for the program should present carefully prepared abstracts of about 200 words which should be submitted in sufficient time to permit of their publication before the meeting so that they may be distributed at the opening session. No descriptions or mention by name of new species should be included in these abstracts.

The Business Manager of PHYTOPATHOLOGY presented his report and accounts. The accounts were audited by a committee consisting of W. A. Orton and F. C. Stewart, and approved. The account showed a small balance on hand after all debts connected with the publication of the Journal were paid.

The following editors were elected: L. R. Jones, C. L. Shear, and R. A. Harper, in place of H. H. Whetzel, resigned. The associate editors were all reëlected, with the exception of R. E. Smith, who resigned. C. J. Humphrey was elected to fill the vacancy. The Secretary was instructed to divide the associate editors into four

groups of three each: the first group to hold office four years, the second group three years, the third group two years, and the fourth group one. This arrangement to be determined by lot.

A motion was adopted making available for the use of the Business Manager in connection with PHYTOPATHOLOGY during the coming year $200, or such portion thereof as may be necessary, from the funds of the Society.

The Treasurer presented the following report, which was examined and approved by the Auditing Committee:

RECEIPTS

Jan. 1	Cash balance from 1911	$218.44
Dec. 30	232 membership dues	696.00
Nov. 1	Interest on bank deposit, 1912	8.33
	Total	$922.77

DISBURSEMENTS

Dec. 30	232 membership subscription at $2	$464.00
	Stamps, stationery, typewriting, and stenographic work	17.23
	Printing circulars, etc	6.25
	Traveling expenses, Secretary, Cleveland	27.00
	Total	$514.00
	Balance on hand in bank	408.29

The phytopathological exhibit was a very interesting and attractive feature of the meeting.

A symposium on International Phytopathological Problems was held on Thursday and attended by members of Section G of the A. A. A. S., and also the Botanical Society of America.

The following papers were presented at this symposium. Most of these papers will be published in PHYTOPATHOLOGY this year.

The importance of closer international relations in phytopathology. L. R. JONES

Some personal observations on phytopathological problems in Europe and America. C. L. SHEAR

Theory and practice of legislation against plant diseases. H. T. GÜSSOW

International phytopathological problems connected with quarantine legislation. W. A. ORTON

Abstracts of the other papers presented, so far as they have been received, follow.

Effect of the steam-formalin treatment on certain soil organisms. J. R. WINSTON

No abstract.

A rot of grapes caused by Cryptosporella viticola. C. F. GREGORY

Published in full in this issue.

Notes on the genus Endothia. H. W. ANDERSON

A brief summary of the literature dealing with this genus is given, together with a discussion of the species definitely established and the forms recently investigated. The reasons for including the chestnut blight fungus in this genus, and a discussion of *Endothia parryi* (Farl.) follow. The production of pigment by the various species in culture, and its significance, are considered and the formation of the pycnidia and conidia in *E. parasitica* and *E. virginiana* is outlined.

Wind dissemination of the chestnut blight organism. PAUL J. ANDERSON

The ascospore stage of this fungus was found maturing in abundance during every month of the summer. The ascospores are ejected into the air from the ostioles during rain, and as long afterward as the bark remains wet. A pustule may eject spores continuously for three weeks if kept wet. The distance to which the spores are shot and the rate of ejection were studied. Tests were made of the spore content of the air by the use of an aspirator and by exposure of sterile plates. By the latter method spores were caught at a distance of 51 feet. The average of a number of aspirator tests gave 4.3 spores per liter of air. Successful inoculations were made by cutting wounds and permitting spores to shoot into them from bark held at various distances. The writer believes that the wind is the most important agent in the spread of the disease.

Some notes on the dissemination of Diaporthe parasitica. F. D. HEALD

No abstract.

Fusarium batatatis Woll. MS., not Nectria ipomaeae Hals., the cause of the sweet potato stem rot. L. L. HARTER and ETHEL C. FIELD

For many years *Nectria ipomaeae* Hals. has been regarded as the cause of the sweet potato stem rot. Numerous inoculations, both in the greenhouse and in the field, with conidia and ascospores of *Nectria ipomaeae*, however, have failed to produce the disease. In the greenhouse, where temperatures and relative humidity were high, some of the plants would show considerable injury and enlargement of the wound about the point of inoculation, and perithecia were sometimes formed in the injured areas. The organism, however, never entered the bundles of the stem, and several attempts to isolate it from the living tissue of the plant have always been unsuccessful. When field inoculations were made, the wounds where the spores were inserted healed normally and no injury to the plant resulted.

Another organism, soon to be described by Dr. H. W. Wollenweber as *Fusarium batatatis* has frequently been isolated from the fibro-vascular bundles of the roots, petioles and stem. This has been found to be the real cause of stem rot. A large number of inoculations have been made in the greenhouse and in the field and a large percentage of the plants showed the characteristic symptoms of the disease. The organism has been recovered from the fibro-vascular bundles of the roots, stems and leaves of many inoculated plants, in many cases from the stem 2 to 3 feet from the point of inoculation. A detailed study of the disease is soon to be published.

Studies on the water core of apple. P. J. O'GARA

Water core is a trouble which is found wherever apples are grown. It has been reported from all parts of the United States, as well as from Europe, Asia and Africa. It is found more particularly in districts where very great ranges of temperature and relative humidity are experienced. It has been found that the addition of water to orchards, either naturally or artificially, when followed by extremely high maximum temperatures with low humidity during hours of sunshine, and low temperatures with high humidity during the night, will induce water core. Young trees bearing their first crops are more susceptible than older, less vigorous apple trees. Water-cored fruits are found mostly on the terminals and on the south and southwest sides of trees, because such fruits are subject to greater extremes of temperature. The side of a fruit presented to the direct rays of the sun will show more water core than the opposite side. Heavily pruned trees, or trees defoliated shortly before the ripening period begins are very liable to water core if climatic conditions are favorable to the disease.

Moderately water-cored fruits if placed in storage at an even temperature will recover, the water-soaked spots entirely disappearing. Fruits in which the seed cavities have become filled with liquid will not recover. It has been demonstrated beyond doubt by inoculation experiments that water core is not due to the attack of any parasite.

The apothecial stage of Sclerotinia fructigena (Pers.) Schroet. in Ontario in 1912. J. E. HOWITT

No abstract.

Leaf roll, curly leaf and other new potato diseases. W. A. ORTON

In treating of the diagnosis of diseases hitherto confused, differentiating characters were pointed out for the following troubles:

1. Fusarium wilt, an American disease not yet proved to occur in Europe. Characterized by wilting and early death of the plant, and browning of the vascular tissues of stem, stolon and tuber, which are infected by *Fusarium oxysporum* (Schlecht.) Sm. and Sw.

2. Verticillium wilt, a disease found in America and Europe. Resembles the foregoing, but is associated with the fungus *Verticillium albo-atrum*. Wilting is usually more rapid, vascular browning darker, and dead stalks quickly covered with grey layer of spores.

3. Leaf roll (Blattrollkrankheit), a trouble of unknown nature, probably non-parasitic, characterized by upward rolling of the leaves, a decreased yield of tubers and by hereditary transmission of the diseased conditions. The extensive occurrence of this disease in Colorado and western Nebraska was reported, and it was held responsible for the greater part of the heavy crop losses there in 1911 and 1912.

4. Curly leaf (Kräuselkrankheit), an inheritable, physiological derangement of the potato plant, resulting in dwarfed growth, decreased yield and, particularly, in a shortening of all vascular parts, the stem, branches, petioles and midribs. The leaves are crinkled and curled. Common in Europe and the eastern United States.

5. Rosette, aërial tubers and other troubles distinct from the foregoing and associated with Rhizoctonia lesions on stem, roots and stolons.

The following new potato diseases were described for the first time:

6. Mosaic, apparently similar in nature to the mosaic of tobacco, tomato, etc.

7. Streak. Elongated or angular spots appear on leaves, following the veins. The leaves wither and hang dry on the plant, which soon dies. Cause unknown.

Potato tuber rots associated with Fusarium were also differentiated. The full text will shortly appear as a bulletin of the Bureau of Plant Industry.

Notes on some western Uredineae which attack forest trees. II. G. G. HEDGCOCK

Read by title. Published in full in this issue.

Notes on winter-killing and smelter injury in the forests of Montana. II. G. G. HEDGCOCK

Read by title.

Black pit of lemon. CLAYTON O. SMITH

A bacterial disease of lemons, causing a brownish colored depressed pit on tree-ripe fruit in the orchard, is described. A pathogenic organism, *Bacterium citriputeale*. nov. sp., has been isolated and its cultural characteristic studied. Successful artificial puncture inoculations have been made on lemons, limes, oranges and grape fruit.

The American botrytis blight of peonies. H. H. WHETZEL and J. ROSENBAUM

The peony is generally held to be a perennial especially free from diseases. It is, however, subject to several, the most destructive of which appears to be the one here under consideration.

Halsted first applied the name "mould of peonies" to this disease, but in order to specify its causal character and at the same time distinguish it from a similar disease in Europe the name "American Botrytis blight" is proposed.

Halsted who was the first to record the disease, regards it as epidemic in eastern United States in 1897. It is now more or less destructive throughout this section every rainy spring.

The most striking symptoms are the rotting of the stems at the base soon after they are up in the spring, followed later by the blasting and rotting of the unopened buds and the blighting of the leaves.

The disease is caused by a Botrytis. The pathogenicity of the fungus has been fully established by two series of successful inoculation experiments and subsequent reisolations. The identity of the pathogen has not yet been satisfactorily determined but the evidence indicates that it is not identical with *Botrytis paeoniae* Oud., reported by J. Ritzema Bos as a destructive disease of peonies in Holland and Germany, and noted by Massee as destructive to the same host in England and Ireland. Halsted held it to be but a virulent form of *Botrytis vulgaris*.

The parasite passes the winter in sclerotial form in the old stems or stubble. From these conidiophores bearing conidia for primary infections arise in the spring. Ants appear to be important agents in carrying the conidia to the buds. No method of control has as yet been demonstrated.

Resting mycelia of Phytophthora and other related species. I. E. MELHUS

It has been shown by careful experiments that the mycelium in Phytophthora infected tubers can spread from the tuber into the sprouts when placed in a warm, saturated atmosphere, and that the stems of plants growing from such tubers may become infected from below upward. Such diseased plants are produced more readily in a saturated atmosphere at a comparatively high temperature.

It has also been demonstrated that the mycelium can function in a similar capacity in other species closely related to Phytophthora, namely, *Peronospora parasitica* on *Lepidium viriginicum*, *Peronospora ficariae* on *Ranunculus fascicularis*, *Plasmopara halstedii* on *Helianthus divaricatus*, *Cystopus candidus* on *Capsella bursa-pastoris and Lepidium viriginicum*.

Some rose anthracnoses. JOHN L. SHELDON

A report is given of some anthracnoses of wild and cultivated roses, one of which was rather serious in certain localities. It was found that this one could be controlled by removing the infected canes early in the spring. Asco-stages of each of the fungi were obtained; one of them resembled species of Gnomoniella while the others were more like what is commonly known as Glomerella.

The diseases of the sweet pea. J. J. TAUBENHAUS

This paper presents the results of further observations on the sweet pea anthracnose (*Glomerella rufomaculans* (B) Sp. & V. Sch.). The mosaic disease, and damping-off diseases due to Rhizoctonia and *Sclerotinia libertiana* are recorded for the first time as attacking sweet peas under glass, as well as out of doors. *Thielavia basicola* is recorded for the first time in this country as causing a root rot of sweet peas. A mildew disease of the sweet pea (*Erysiphe polygoni*?) is also recorded as troublesome to sweet peas under glass.

The black rots of the sweet potato. J. J. TAUBENHAUS

In describing the black rot (*Sphaeronema fimbriatum* (E. & H.) Sacc.) of the

sweet potato B. D. Halsted also recorded a Sclerotium stage of that fungus which invades the entire roots and reduces it to a charcoal. Careful observations and studies by the writer have definitely demonstrated that the Sclerotium fungus is not a stage of *Sphaeronema fimbriatum* but that it is distinct from the latter, and the name *Sclerotium bataticola* n. sp. is proposed, together with the name "charcoal disease." This rot may often attack a root which was previously infected by *Sphaeronema fimbriatum* or it may attack healthy roots independent of the latter fungus. A third rot due to Lasiodiplodia is also recorded and *Lasiodiplodia tubericola* was found to be an active parasite on sweet potatoes.

The possibilities of disease resistance in cabbage. L. R. JONES

The cabbage, under intensive and continual culture, suffers peculiarly from plant parasites which persist in the soil. Club root (Plasmodiophora), black rot (Pseudomonas), black leg (Phoma), and yellows (Fusarium) are examples of such which are combining to make cabbage culture unprofitable over large areas of high-priced land in southern Wisconsin. Of these diseases yellows is the worst. Experiments during the last three years have failed to discover any specific remedy. On the other hand, the possibilities of developing disease resistant strains are encouraging. Trials of the last two seasons show certain standard varieties to have some resistance, especially the Houser, but Wisconsin growers do not like this as a commercial type. In even the worst-diseased fields occasional plants escape and mature apparently healthy heads.

About 100 such plants were selected in 1910 from 3 different fields, representing different strains of Danish ball-head varieties of winter cabbage, using care to select the best commercial types. Seed was grown from these in 1911 and tested in 1912 on soil badly infected with the yellows disease. The progeny of every one of these selected heads showed a high degree of disease resistance. The *average of all* of the heads for each of the three strains was as follows: Strain W, 70 per cent lived, 44 per cent headed; Strain H, 86 per cent lived, 53 per cent headed; Strain B, 93 per cent lived, 68 per cent headed. In contrast with these, the *best* of the commercial strains of the various varieties tested, including the Houser, gave only 27 per cent heads. The above figures are averages of *all* resistant strains; the *best* of these made an even better showing, viz., 98 per cent lived and 93 per cent headed. These had all been selected as showing desirable commercial characters and they reproduced these true to type. Heads of the best of these strains have been saved and from them an abundance of seed should be secured next year for further trial and possible selection.

Mycosphaerella pinodes the ascigerous stage of Ascochyta pisi. R. E. VAUGHAN

Ascochyta pisi Lib. is associated with *Septoria pisi* West. and other fungi in causing serious damage to the pea crop of Wisconsin. *Mycosphaerella pinodes* (B. & Bl.) Johanns. was observed in the autumn of 1911 and 1912 on dead vines previously attacked by Ascochyta. In one case Mycosphaerella developed in the greenhouse on plants inoculated with Ascochyta.

On October 4, 1912, mature ascospores were obtained from Mycosphaerella perithecia on dead pea stems. Several of these were crushed in water and the spores stippled on leaves of a healthy pea plant which was then kept in moist atmosphere in the greenhouse. After five days characteristic brown lesions appeared, resembling in every way those caused by *Ascochyta pisi*. In ten days pycnidia with mature Ascochyta spores were obtained. This experiment has been repeated several times with Mycosphaerella spores from different parts of Wisconsin, with the same result in each case.

A single Mycosphaerella spore was removed from a drop of 5 per cent gelatin solution, by means of the Barber spore-picking apparatus, and transferred to synthetic

agar. The development from this spore was observed from day to day, and on the seventh day from inoculation the culture was found to have spores of *Ascochyta pisi*. In order to prove that these spores were capable of producing lesions on the pea plant, a suspension was sprayed over healthy plants, which were then placed in moist atmosphere. In this case, also lesions and pycnidia of Ascochyta were produced, while control plants remained normal.

These observations and experiments show that *Mycosphaerella pinodes* is the ascigerous stage of *Ascochyta pisi*.

Observations on the migration of Bacillus amylovorus in the host tissues. FREDA M. BACHMANN

Published in full in this number.

Some successful inoculations with the peach crown gall organism, and certain observations of retarded gall formation. CLAYTON O. SMITH

Reference is made to the work of Dr. Erwin F. Smith and his associates in establishing a wide range of hosts for *Bacterium tumefaciens*. Certain hosts not previously reported as susceptible have been successfully inoculated at the Whittier (California) laboratory. A list of plants successfully inoculated is given. Thirty-five species and a number of varieties, representing eight families, are listed. Retarded gall formation is described on Angiers quince and on fig. The organism may live for as much as a year and a half within the tissues of the host without causing evident gall formation.

A botrytis disease of dahlias. MEL. T. COOK

The fungus attacks the roots in storage, causing a rot, and is especially severe in moist places, but is of no importance if the storage houses are reasonably dry and well-ventilated. When the rotting roots or cultures are allowed to dry gradually, the fungus produces sclerotia of various sizes. No ascospore stage has been found.

Conditions influencing infection of apple leaves by Gymnosporangium macropus. H. R. FULTON

Inoculation tests and observation on natural infection indicate that the individual leaf in its growth passes from a condition of immunity, due to its very young condition, to one of maximum susceptibility as it reaches something more than half its full size; with increasing age it becomes less susceptible, and finally immune. On twigs the infection occurs in zones of usually three to five infected leaves, in which there is a decrease in the amount of infection from the intermediate leaves upward and downward to those respectively younger and older at the time of infection.

Alternaria rot of apples. MEL. T. COOK and G. W. MARTIN

This fungus attacks several varieties of apples, but is most severe on the Jonathan. It gains admission to the fruit through the lenticels and causes small, shallow, dry rot spots. It fruits abundantly in culture, producing very characteristic spores. It is different from the blossom-end and core rot reported by Longyear from Colorado in 1905, and from the blossom-end and core rots found in New Jersey. The spores of the organism show numerous papilla-like markings.

The use of the green muscardine in the control of some pests of sugar cane. JAMES BIRCH RORER

To be published in PHYTOPATHOLOGY soon.

A disease of peanut plants caused by Bacterium solanacearum. H. R. FULTON and J. R. WINSTON

The identity of the causal organism with the one producing bacterial wilt of tobacco was established by cross inoculations with strains of the Bacterium from

the two hosts, by reisolation and later reinfection of each host, and by comparison of cultural and morphological features and staining reactions.

Control of apple rust by spraying. N. J. GIDDINGS and D. C. NEAL

Published in last number of PHYTOPATHOLOGY.

Further cultures of heteroecious rusts. W. P. FRASER

This paper describes field observations and artificial infection experiments which show that five of the fern rusts belonging to the genus Uredinopsis are heteroecious, having their aecial stage on *Abies balsamea* (L.) Mill., the aecia being the white spored forms that have passed as *Peridermium balsameum* Peck. The species thus connected were:—*Uredinopsis struthiopteridis* Stormer, *U. osmundae* Magn., *U. phegopteridis* Arthur, *U. atkinsonii* Magn. and *U. mirabilis* (Peck) Arthur. The paper also describes infection experiments with *Pucciniastrum myrtilli* (Schum.) Arthur, which infected *Tsuga canadensis* (L.) Carr., thus confirming the life history as previously established by Dr. Clinton. *Melampsora arctica* Rostr. was also successfully sown on *Abies balsamea*, and *Melampsora medusae* Thum. was likewise successful on *Tsuga canadensis*, confirming previous work of the writer.

Some field experiments with the chestnut canker fungus. W. H. RANKIN

The average rate of growth of artificially produced cankers during the summer months was 1.88 cm. per month (four weeks). The conidia appeared about one month after inoculation. The perithecial stromata however were not formed until late in the summer even in the case of infections produced in the spring. Mature perithecia were common by the middle of November in all cankers produced by inoculations made even as late as August 1. Ascospores are ejected only when free water is present on the stromata. Mature ascospores can be found at any time during the year. They are shot out in vast numbers with every rain during the summer and are carried by the wind. Ascospores yielded 100 per cent infections. The water content of the tree, as observed in 1912, does not alter the susceptibility of the trees to the fungus. Slow growing cankers can be produced on certain species of oaks. *Endothia virginiana* Anders. & Anders. is not pathogenic on chestnut in New York State.

Cronartium ribicola and the proscription of Ribes nigrum. F. C. STEWART and W. H. RANKIN

Another severe outbreak of Cronartium on currants occurred at Geneva, N. Y., in 1912. Six nurseries and ten fruit gardens were affected. In one nursery almost every leaf was affected on 15,000 black currant plants. Apparently, the disease is established at Geneva, although no specimen of the Peridermium stage on pine has yet been found there. Probably, it is also established at other places in New York and in Massachusetts and Connecticut. Complete eradication of the disease is no longer possible.

Observations made during the Geneva epidemic convince the writers that in attempts to control the pine blister rust vigorous warfare should be waged against the black currant. Owing to its great susceptibility and its habit of holding its leaves until late in the season, the black currant is particularly dangerous as an agent in the spread of the disease. In the uredinial stage, *Cronartium ribicola* readily spreads from one black currant plantation to another over distances of 800 meters or more. State authorities should discourage the further planting of black currants by seeking out diseased plants and promptly destroying them. This policy will be pursued in New York. Compared with the white pine, the black currant is of small consequence.

Effect of the steam-formalin treatment on certain soil organisms. J. R. WINSTON

Soil infected with Fusarium from tomato, and Rhizoctonia from potato, was given the steam-formalin treatment, and soil with similar infection was steamed to determine the efficiency of the steam-formalin treatment. When the steam-formalin treatment was used, pathogenic organisms were killed in about one-half the time required by steam alone. Tomato plants were grown in the above treated soil to determine the presence or absence of the pathogenic fungi.

The small lettuce Sclerotinia, an undescribed species. IVAN C. JAGGER

In 1900 a fungus similar to *Sclerotinia libertiana* Fuckel, but producing much smaller sclerotia, was described by Smith from Massachusetts greenhouse lettuce. He concluded that the fungus was a form of *S. libertiana* which has lost the ability to produce the perfect stage. The characteristic small sclerotia have been grown in pure culture and from them have developed small apothecia. The disc of the apothecium is lined with a layer of 8-spored asci interspersed with paraphyses. The ascospores germinate readily on nutrient media producing cultures with small sclerotia, and having other characters identical with the original. The fungus appears to be an undescribed species of Sclerotinia clearly distinct from *S. libertiana*.

Agar culture of wheat as a means of seedling purification. D. G. MILBRATH

This paper is a partial report upon certain investigations upon root diseases of wheat conducted in the laboratory and in the experimental fields of the North Dakota Agricultural College during the years 1910 to 1912. The work is a continuation of similar work undertaken and directed by Professor Bolley during the previous years of 1908 and 1909. The purpose of the work is to ascertain, by pure cultures, the possibility of purifying seedlings of wheat or other cereals so that physiological, or other experiments, may be carried on with wheat plants known to be internally free from disease-producing organisms.

The Thielavia disease of violets. DONALD REDDICK

Thielavia basicola Zopf has been found doing very serious damage to violets in commercial and private houses. The roots become infected and are rotted off at the point of attack. The occurrence of the disease on the roots is usually manifest by the peculiar yellowing and slight curling of the foliage.

Runners and leaf petioles become infected abundantly and in this condition the disease is spoken of by growers as the black rot disease. When infected runners are used for cuttings the fungus is apt to spread to the young roots. As high as 50 per cent of the plants as they come from the sand may be thrown away on this account. Plants grown from diseased cuttings are dwarfed and bear an inferior quality of flowers, although the quantity may be even greater than from healthy plants.

A culture of the fungus obtained from a single germinating segment of a chlamydospore has been used for inoculation purposes and the disease has been produced by artificial inoculation, the period of incubation being from ten to twelve days. Chlamydospores are to be found developed externally, but in old lesions they can be found densely packed in the cortical cells or even in the cells of the fibro-vascular bundles.

Physoderma zeae-maydis Shaw in Illinois. J. T. BARRETT

No abstract.

A bacterial disease of the sweet pea and clovers. THOS. F. MANNS

A disease of the sweet pea in England popularly known as "streak," and which heretofore has been assigned to different causes, is demonstrated to be bacterial.

It is related to a disease of clovers and apparently of other legumes. Cross inoculation work has been positive between sweet peas and red clover, also infections have been produced on red clover with the organism from the different clovers, soy bean, several Lathyrus species and other hosts. The disease is characterized on the sweet pea by brown spots or streaks on the stems which kill the cambium. On the clovers the stems are blackened and the leaves spotted, like bacteriosis of beans. Infection is by way of the stomatic openings. Extensive cultural and biological studies indicate this to be a new species to which is applied the name *Bacillus lathyri* Manns & Taubenhaus.

Notes on some Nebraska potato diseases. E. MEAD WILCOX

No abstract.

Some laboratory conveniences for the pathologist. E. MEAD WILCOX

No abstract.

The brown-rot canker of the peach. R. A. JEHLE

The brown-rot fungus *Sclerotinia "fructigena"* was found to be the cause of cankers on the limbs of peach trees. At first the lesions are slight depressions but soon become open wounds with copious gum flow. Later they become black and rough. The lesion increases in size from year to year. The cankers may be formed by the fungus growing back from a brown-rotted fruit through the fruit spur into the limb but ascosporic infection of blossoms is thought to be the greater source of trouble.

Sclerotinia "fructigena" from several sources in the United States was used for inoculation purposes. Infection has occurred in all of the two hundred or more inoculations thus far made on limbs of all ages.

Notes on the fungus diseases of sugar cane in Porto Rico. JOHN R. JOHNSTON

The early literature on the sugar cane fungi of Porto Rico is very indefinite as to their identity. The writer has collected many fungi on cane, and some of the most important ones have been identified by experts. A list of the fungi is given, together with notes as to their importance.

Marasmius sacchari and not *M. plicatus*, is stated to be the most common sugar cane agaric in Porto Rico. *M. sacchari* was positively identified on living cane, on cane trash, on *Panicum barbinode* and on dead tissues of *Bromelia pinguin*.

Colletotrichum falcatum is found not only on cane, but apparently also on dead petioles of *Carica papaya*, accompanying another species of Colletotrichum. *Thielaviopsis paradoxa* causes a disease both of the sugar cane and of pineapples in Porto Rico. *Thyridaria tarda*, *Nectria laurentiana* and *Spegazzinia ornata* are reported both on cane and other plants.

Melanconium sacchari and *M. saccharinum* are reported as common. Besides the preceding, practically all the important cane fungi of other countries are also reported. The important disease-producing organisms not yet found in Porto Rico are the cane smut, the rust, and the organism causing the yellow gumming disease.

The diagnosis of wilt diseases and the diagnosis of Ascomycetes from their conidial stage. H. W. WOLLENWEBER

Published in this number.

Helminthosporium diseases of barley in Wisconsin A. G. JOHNSON

Three distinct diseases of barley, caused by as many species of Helminthosporium, were observed in Wisconsin during the past season. By the symptoms and characters of the causal fungi, these may be segregated as follows:

A. Light yellow to brown striae in the leaves; infection systemic.

1. Stripe disease caused by *H. gramineum* Rabh.

B. Oval to oblong brown blotches in leaves; infection local.

2. European blotch disease caused by *H. teres* Sacc. (Spores cylindrical, rounded at ends, usually straight).

3. American blotch disease caused by *H. sativum* P.K.B. (Spores narrowly spindle-shaped, usually more or less curved).

Damping-off and root rot parasites of sugar beets. H. A. EDSON

This paper was a report of progress on work which contemplates a comprehensive study of the seedling and root rot diseases of sugar beets throughout the country. By carefully controlled inoculation experiments with pure cultures, four fungi have been shown to stand in causal relation to seedling troubles. These are *Pythium de baryanum* Hesse, *Aphanomyces levis* de Bary, *Phoma betae* Frank and a species of Rhizoctonia probably identical with the form described as *Corticium vagum* B. & C. var. *solani* Burt, though the perfect stage has not been observed. Two of these fungi, Phoma and Rhizoctonia, are also capable of producing decay in mature beets.

Phoma betae was invariably present on beet seed examined, but so far has not been isolated from the soil. The other species mentioned appear to be soil borne and were not found on seed. For the proper control of inoculation experiments with seedlings some form of seed sterilization is necessary. Among the chemicals tried with negative results are, hydrogen peroxid, hydrochloric acid up to full concentration for fifteen minutes, followed by lime water, sulphuric acid up to full concentration for one hour, followed by lime water, and formalin solution up to 2 per cent for various intervals up to one hour. A more satisfactory method which was employed in all the inoculation work consists of pasteurization as suggested by Peters.[1] The seed was soaked in water at 60° C. for ten minutes, cooled in water, dried on filter paper and after an interval of twenty-four hours again heated for ten minutes in water at 60° C., after which it was placed in sterilized soil and watered with disease-free water. Numerous strains of the several fungi secured from various sources were employed. Those enumerated were invariably recovered from the diseased seedlings obtained by inoculation and passed through from four to six additional generations of seedlings.

The stem rot or the Hawaiian "Iliau" disease of sugar cane. C. W. EDGERTON

Read by title.

To be published in Phytopathology soon.

Hop mildew in New York. F. M. BLODGETT

This disease, caused by *Sphaerotheca humuli*, has been destructive in the hop yards of New York State for the past four years. Its control was undertaken by means of dusting with flowers of sulphur. Although the mean temperature of New York State for the summer months is considerably below that at which sulphur is said to be effective, very satisfactory results have been secured even under the unusually cool conditions prevailing during the season of 1912.

Powdery or corky scab, Spongospora subterranea (Wallr.) Johns. and its occurence in North America. H. T. GÜSSOW AND J. W. EASTHAM.

Published in full in this number.

C. L. SHEAR.
Secretary-Treasurer.

[1] Über die Erreger des Wurzelbrandes. Arb. a. d. Kais. Biol. Anst. f. Land- u. Forstwirtschaft **8**, Heft 2.

[PHYTOPATHOLOGY for December, 1912 (**2**: 217-276, *pls.* XXI–XVI was issued December 28, 1912.]